VETERINARY MEDICINE

OCTOBER 1939

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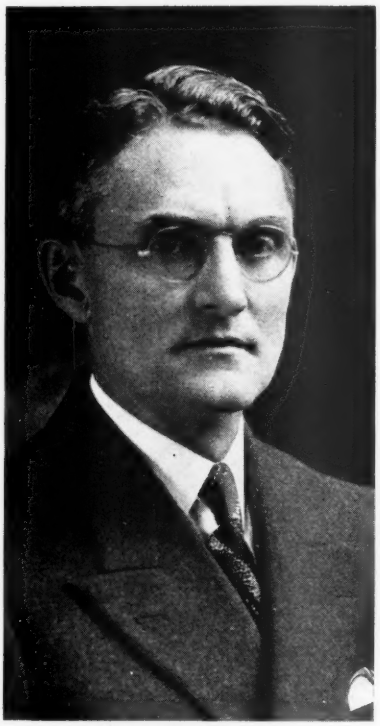
Memphis Meeting of the A. V. M. A.



Henry Dale Bergman
President of the A. V. M. A. 1938-39

The 76th Annual Meeting of the American Veterinary Medical Association, held in Memphis, August 28-September 1, 1939, was by all criteria a successful meeting. The weather was agreeable, the arrangements were excellent and the attendance exceeded the highest estimates of veteran guessers. Subject to some correction from rechecking, the number who registered at the meeting may be said to be in excess of 1500. This places the Memphis meeting in attendance second only to the Columbus meeting in 1936 and far ahead of any meeting not held in a center of veterinary population.

An adequate hotel headquarters adds greatly to the success and enjoyment of any meeting and the headquarters hotel at Memphis proved adequate—almost. The banquet exceeded the capacity of the facilities by some 50 or 60, and there were insufficient rooms for the banquets of the alumni associations, in a few instances two and in one instance three alumni associations being grouped together. However, little objection can be raised to matters of this kind. There are probably not more than half a dozen cities in the country that contain hotels wholly adequate for a large A. V. M. A. meeting and the Association mem-

OPENING SESSION, 76TH ANNUAL MEETING OF THE A. V. M. A., MEMPHIS, AUGUST 28, 1939

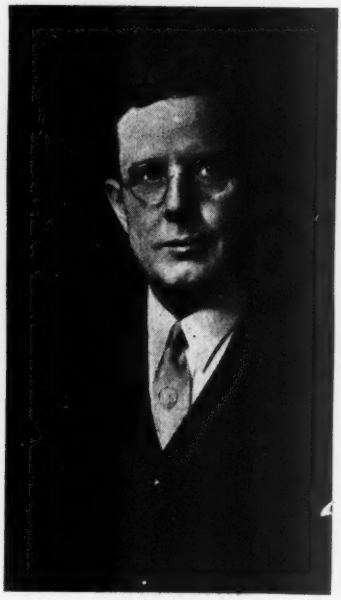
Those seated at the table (left to right) are: Oscar V. Brumley, Columbus, O., President A. V. M. A. 1937-38; Moses Jacob, Knoxville, Tenn., Treasurer 1918-40; Nelson S. Mayo, Highland Park, Ill., Secretary 1913-15, 1918-22; Cassius Way, New York City, President 1939-40; Henry D. Bergman, Ames, Iowa, President 1938-39; Harry W. Jakeman, Boston, Mass., Chairman of the Executive Board 1936-40; and Louis A. Merillat, Chicago, Executive Secretary 1938-40.

bership would not want to meet always in one of them.

The president's address at the opening of the convention was a thoughtful one, and well received by the audience. The presentation of golden keys to the president and president-elect and the awarding of the 13th International Veterinary Congress prize to John R. Mohler, Chief of the Bureau of Animal Industry, U. S. Department of Agriculture, was an impressive ceremony. The presentations were made by H. W. Jakeman, Chairman of the Executive Board.

John Robbins Mohler

John Robbins Mohler was born in Philadelphia in 1875 and was graduated from the School of Veterinary Medicine of the University of Pennsylvania in 1896. He engaged in practice for a year and then entered the Bureau of Animal Industry, which he has served in the capacities of assistant inspector (1897-99), assistant pathologist (1899-1901), Chief of the Pathological Division (1902-14), Assistant Chief of the Bureau (1914-17), and Chief since December, 1917. He has been awarded honorary degrees of D.Sc. by the University of Pennsylvania (1925), Iowa State College (1920) and the University of Maryland (1928). He is an honorary M. R. C. V. S., and at the XIII International Veterinary Congress in Zurich-Interlaken, Switzerland, in 1938 he was made an honorary Life Member. He has been a member of the A. V.

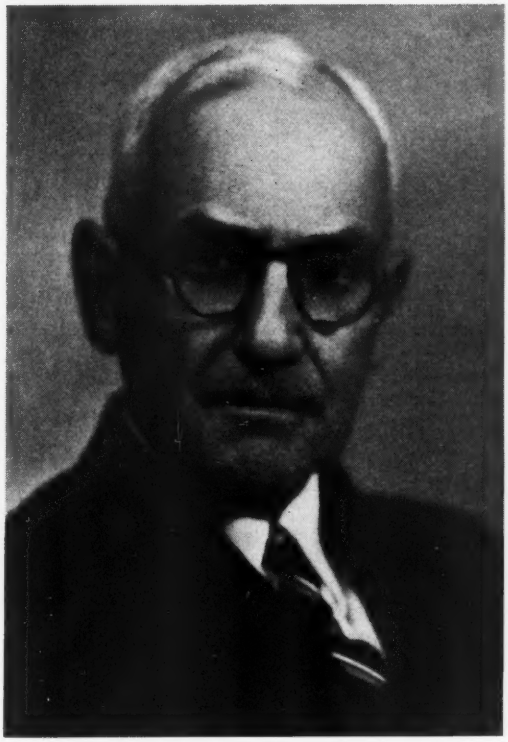


Cassius Way, New York City
President of the A. V. M. A.

Doctor Way studied agronomy and animal husbandry at Connecticut Agricultural College, Storrs, Connecticut, graduating in 1899. In 1905 he received an A.B. degree from Cornell University, and in 1907 the degree of D.V.M. from the New York State Veterinary College. From 1908 to 1919 he was employed by The Borden Company, during the last five years as chief veterinarian, with headquarters in Chicago. He resigned this position to enter general practice, in which he has since been engaged, together with commercial veterinary supply work. He has given special attention to equine practice, particularly the ailments of horses incidental to breeding and reproduction. He numbers among his clients many of the owners of the most valuable thorobred and trotting horses and of the finest horse breeding establishments in the United States. A member of the A. V. M. A. since 1908, he has served on the Executive Board for several years, and was Chairman of the Committee on Local Arrangements for the Diamond Jubilee meeting in New York last year, at which he was chosen President-Elect. Since then he has attended veterinary meetings in various parts of the country and extended his already wide acquaintance. At the close of the Memphis meeting he was installed as President for the year 1939-40.

Doctor Wight was born at Medfield, Mass., April 14, 1876, attending Medfield High School three years, Dean Academy, Franklin, Mass., two years, and entered Harvard Veterinary College in 1894, graduating in 1897. A summary of his activities since that time includes: general practice in Medfield and vicinity until appointed in the Meat Inspection service of the Bureau of Animal Industry, U. S. Department of Agriculture, Boston, Mass., December 15, 1898; assigned to foot-and-mouth disease work, Boston, in February, 1903; transferred to scabies eradication work in South Dakota and North Dakota in October, 1903; returned to the Meat Inspection service at Boston in September, 1906; transferred to Tick Eradication work in May, 1908, with headquarters at Raleigh, N. C.; transferred to foot-and-mouth disease eradication at Danville, Pa., in October, 1908; transferred to the position of inspector in charge of Tick Eradication work in the States of Arkansas, Louisiana and Missouri, with headquarters at Newport, Ark., in June, 1909, headquarters for this work being changed to Little Rock, Ark., in March, 1910; transferred to the Tuberculosis Eradication Division, Washington, D. C., in December, 1917, and succeeded the late John A. Kiernan as Chief of that Division in February, 1928, which position he still occupies.

Doctor Wight is a member of the U. S. Livestock Sanitary Association, which he served as president during 1930, of the National Association of Bureau of Animal Industry Veterinarians, and of the District of Columbia Veterinary Medical Association. He has been a member of the A. V. M. A. since 1913. At the Memphis meeting he was chosen President-Elect, which means that he will serve the Association as president during the year 1940-41.



Alexander E. Wight, Washington, D. C.
President-Elect of the A. V. M. A.

M. A. since 1898 and was president during its Golden Jubilee year (1912-13) and president of the U. S. Live Stock Sanitary Association in 1925.

Doctor Mohler's contribution to veterinary literature have been voluminous. He was translator and editor of Edelmann's "Meat Hygiene" (1908); co-translator of Hutyra and Marek's "Special Pathology and Therapeutics" (1912), Ernst's "Milk Hygiene" (1914), and collaborating editor of Hutyra, Marek and Manninger's "Special Pathology and Therapeutics" (1938). He has written numerous bulletins and other reports of

Louis A. Merillat
Executive Secretary of the A. V. M. A. received unstinted praise for the business-like management of the affairs of the association and for the unfailing courtesy shown all who had business with the secretary's office during the meeting.

the B. A. I., and contributed a large number of articles to veterinary periodicals.

The period of Doctor Mohler's incumbency as Chief of the Bureau of Animal Industry has been signalized by the tuberculosis eradication program undertaken in 1918 and now nearly completed, by great progress in fever tick eradication, which is now complete except for a few counties in Florida and Texas, by the cattle-reduction-Bang's-disease-control program, which later became the Bang's disease eradication project, and which at the present time includes about 12 million animals under supervision, and by a vast amount of veterinary research, both of which (the animal disease control program and the research program) have contributed enormously to animal production in this country. Foot-and-mouth disease appeared in the country several times since Doctor Mohler became Chief of the Bureau, and in each instance the outbreak has been suppressed without extending beyond the state in which it first appeared.

John H. Gillman
Doctor Gillman, chairman of the Committee on Local Arrangements, was the busiest man in Memphis during Veterinary Week (and, by the way, the Mayor of Memphis designated August 28-September 1 as "Veterinary Week.") Doctor Gillman received many compliments on the arrangements made for the meeting.

When nominations for president-elect were called for, but one name was offered, that of A. E. Wight, of the Tuberculosis Eradication Division of the Bureau of Animal Industry, U. S. Department of Agricul-

Albert T. Kinsley
was presented with a life-size oil painting of himself at the KCVC alumni banquet.

K. C. V. C. FISH FRY AND BARBECUE

The most delightful social feature of the Memphis meeting for those who attended was a fish fry and barbecue given the alumni of the Kansas City Veterinary College by Walter J. Lacy ('06) on his plantation near Clarksdale, Miss. Doctor Lacy's plantation is one of the show places of northern Mississippi. The fish fry and barbecue was attended by 54 from the convention, and was a delight in every respect.

ture. He was placed in nomination by M. Jacob, treasurer. Doctor Jacob was reëlected treasurer; L. A. Merillat was reappointed executive-secretary, and E. B. Ingmand was reappointed assistant-secretary.

Indianapolis was selected for the place of the 1941 meeting of the A. V. M. A. The 1940 meeting will be held in Washington, D. C., the city selected by the House of Representatives last year.

THE ARMY AT THE CONVENTION

Seventeen veterinary officers of the U. S. Army attended the Memphis meeting. This is the largest number of army officers ever to attend an A. V. M. A. meeting in peace time, exceeding by two the number that attended at Columbus. Those shown in the half-tone are: standing (left to right): Lt. Col. J. A. McCallam, Col. B. E. Seely, Major S. M. Nevin, Major C. S. Greer, Lt. K. H. Willers; sitting, back row (left to right): Col. W. H. Houston, Lt. Col. S. B. Renshaw, Lt. Col. C. M. Cowherd, Lt. Col. F. L. Holycross, Lt. Col. R. T. Seymour, Major J. L. Owens; sitting, front row (left to right): Lt. Col. L. C. Ewen, Lt. Col. N. M. Neate, Col. J. E. Behney, Col. J. D. Derrick, Col. R. A. Kelser, Col. J. R. Underwood.

The secretary's report revealed that a large number of new members were added to the Association during the year, far more than in any of the last 20 years.

Four outstanding developments in veterinary science during the year were presented during the convention: (1) The research of J. W. Patton into the cause of fright disease in dogs; (2) the research of R. G. Green, M.D., into distemper in dogs and the development of a prophylactic vaccine; (3) the research of J. D. Ray into the effect of temperatures within automobiles and in the carrying trunks of automobiles on hog cholera virus, and (4) the studies of L. T. Webster, M.D., on the efficacy of anti-rabies vaccine and the report of Col. R. A. Kelser, Chairman of the Committee on Rabies. Abstracts of the discussions of these new developments are given on the following pages.

Doctor Webster stated that at present we have no adequate test for anti-rabies vaccine and that use in the field is not an adequate criterion, since control animals are not used and the extent of exposure after vaccination is not known. He has attempted to work out a test by the use of Swiss mice; reaching the conclusion that phenolized vaccine is not effective in Swiss mice and that chloroformized vaccine possesses immunizing properties only when injected into the peritoneal cavity. More recently he has been carrying on experiments to determine if these results in Swiss mice are applicable to dogs, and while the experiments are as yet inconclusive, the indications are that they are applicable. It is thought that within the near future a method will be worked out by which anti-rabies vaccine can be quickly tested by the use of Swiss mice and its effect on dogs thus predicted with certainty.

Considerable effort was made to induce the Association to withdraw its approval of anti-rabies vaccine, which was adopted by resolution at the Los Angeles meeting in 1930. The action was not taken, however. A resolution was adopted stating it was the consensus of opinion of the Association that rabies is essentially a veterinary problem and that the control of this disease should be vested in the veterinary profession.

Col. Raymond A. Kelser presented the report of the Committee on Rabies which aroused more discussion than any other subject considered at the meeting.

The commercial and educational exhibits constituted two attractive features of the meeting. Unfortunately, the location of neither was as convenient to access as could have been wished; but the space was adequate, and the exhibits were numerous and excellent. In the half-tone on page 609 we have attempted to show sections of a number of commercial exhibits. The illustrations, however, give little idea of their attractiveness.

John W. Connaway Columbia, Missouri was elected an honorary member in recognition of having been a member of A. V. M. A. for 50 years. Owing to poor health he was not at the meeting.

J. P. Foster Minneapolis, Minn. a regular attendant of A. V. M. A. meetings was much missed. All were grieved by the cause of his absence—protracted illness.

So-Called Hemorrhagic Septicemia[1]

For many years hemorrhagic septicemia has been a handy diagnosis often used when in doubt, especially when there are pulmonary symptoms, sudden deaths and when autopsy shows petechial hemorrhages. However, its prevalence among domesticated mammals has long been a subject of contention.

George R. Fowler

In addition to presenting the paper here abstracted, Doctor Fowler performed several operations in the clinic on the last day of the meeting.

No doubt hemorrhagic septicemia does occur as a primary and destructive disease of animals but it is so rare that we are not justified in diagnosing it without laboratory confirmation. In eight years, with one possible exception, we have failed to get such confirmation although specimens from all suspected cases have been checked.

Although swine plague is still constantly mentioned among prevalent swine diseases it is seldom if ever found in the western corn belt.

Likewise, although cattle and sheep maladies are frequently diagnosed as hemorrhagic septicemia, such diagnoses are rarely confirmed by the laboratories.

The supposedly typical symptoms and lesions of hemorrhagic septicemia can result from a variety of causes such as deficiencies, toxins, viruses, other infections and even the invasion of some parasites. Evidence is accumulating that, when present, the pasteurella organism is a secondary invader.

A diagnosis of hemorrhagic septicemia has long seemed popular with the laity, partly due to the impressive title, and we fear that this plus the ease of administering specific bacterins has made it too popular with the profession. We would plead the case of accuracy by considering hemorrhagic septicemia as innocent until proved guilty.

[1] Abstract of a paper by W. A. Aiken, D. V. M., Merrill, Iowa.

Anesthesia of Large Animals[2]

Anesthesia means without sensation. Some form of anesthesia should be used in all surgery except in those minor operations where the anesthesia will cause more discomfort to the animal than the operation.

Anesthesia is divided into two classes: (1) General anesthesia, characterized by loss of consciousness, and (2) local anesthesia, in which greater or lesser areas of the body are made insensible to pain, but consciousness is not lost.

General anesthesia may be produced by inhalation, in which the animal inhales the vapor of a drug such as chloroform; by injecting the drug into the blood stream; or by placing the drug into the digestive tract, frequently by means of a stomach tube.

The physiology of anesthesia must be understood, as a knowledge of this is necessary so the anesthetist may choose the proper drug and method of administration to suit any particular case. The signs of beginning anesthesia, or surgical anesthesia, and the danger signals must be known to the man who administers the anesthetic.

The various drugs used in general anesthesia, such as chloroform, chloral hydrate, and the barbiturates, must be known and their advantages and disadvantages must be kept in mind at all times. Anesthetic shock, its cause, prevention and treatment is another detail that must be remembered when general anesthetics are employed.

Local anesthesia is produced by injecting into an area of the body a drug which temporarily destroys sensation in the part. The special indications for this form of anesthesia are numerous.

Local anesthesia is divided into two

[2] Abstract of a paper by Geo. R. Fowler, D. V. M., Head of Department of Veterinary Surgery, Iowa State College, Ames, Iowa.

classes: (1) Infiltration, in which the part to be operated is filled with the anesthetic. (2) Nerve blocking, in which anesthetic is injected into or around the nerve supplying sensation to the part. Spinal anesthesia is

Hubert Schmidt

a form of nerve blocking in which the entire spinal cord is blocked, but it is seldom used in surgery of large animals. Epidural anesthesia is a form of nerve blocking in which the nerves are blocked within the spinal canal but after they have left the spinal cord. It is used frequently, especially in cattle.

The site of injection must be carefully prepared to prevent the introduction of infection, and all equipment must be sterile. Different types of needles and syringes are employed for the various occasions.

The principle drugs used in local anesthesia, such as procaine, novacaine, butyn and apothesine, have specific qualities which must be considered in determining which of these drugs to use in any given case. The maximum quantities that may be used with safety for the various species and ages of animals is of great importance to prevent possible disaster. The symptoms and treatment for an overdose must be kept in mind at all times.

The methods of infiltration anesthesia are several and each has a specific use. A knowledge of the location of the chief sensory nerves that supply certain areas which are frequently subject to operation must be well known to the surgeon. Operations are rendered painless by this means.

Treatment and Prevention of Mineral Deficiencies[3]

This discussion is confined to the minerals essential for horses and ruminants which are likely to be deficient in the natural herbage upon which the animals subsist, or which are likely to be deficient in the feed provided for the animal by man; and which, in case of a deficiency, may eventually lead to interference with the well-being of the animal. These include especially phosphorus, calcium, iron, cobalt and iodine.

Through diligent research work in the past the amounts required of those mineral elements by the different classes of animals have been roughly determined. In the case of calcium and phosphorus it has also been determined that these two elements must be present and available to the animal in a fairly definite proportion to one another. This latter fact is very frequently lost sight of and for that reason we frequently find animals suffering from a phosphorus deficiency which reflects itself not only in ill health, but also in poor performance and production to a degree that the economic returns are frequently on the negative side. The amount of these elements available to the animal depends upon the abundance in the ration. These two elements vary widely in the different parts of the plant and especially do we find that the herbaceous part of the plant is the main storehouse for the calcium, while the seed is the main storehouse for the phosphorus. It follows that should the animal receive mostly the herbaceous part it is likely to suffer a phosphorus deficiency and should it be fed mostly on the grain part of the plant it is likely to get too much phosphorus and not enough calcium. But, the calcium and phosphorus content of all plants are not the same, some plants being comparatively low and others comparatively high in calcium and all classes in between. The same is true of the phosphorus content. Nor does the calcium and

[3] Abstract of a paper by H. Schmidt, Dr. Med. Vet., Chief of the Division of Veterinary Science, Texas Agricultural Experiment Station, A. & M. College of Texas, College Station, Texas.

phosphorus content of the plant remain the same throughout its life but it varies so that the young, rapidly growing plant is comparatively rich in these elements while much of them is lost after the plant matures, and there is a further material loss through drying and leaching. These elements are also more readily available to the animal when offered in the form of young, green plants. All of these factors must be considered in the nutrition of farm animals whenever one is dealing with mineral elements in feeds that are on the borderline of sufficiency. That vitamin D is needed for the proper utilization of these elements, especially when they are provided only in borderline amounts, is well known.

Walter R. Krill

An imbalance of these two minerals or a gross deficiency of one or the other or a gross excess of one or the other will cause rickets in the young animal. In the older, more mature animal we speak of the condition as osteomalacia and osteodystrophia fibrosa. Clinically these diseases manifest themselves in unthriftiness, emaciation, lameness, enlargement of bones, poor food consumption, weakness and finally death.

In the case of iron, copper and cobalt, we are dealing with three minerals that profoundly affect the blood picture of the animal and an improper supply of them may lead to marked anemia. Especially prevalent is a cobalt deficiency in certain parts of the world and the clinical condition caused by it has usually received a local name, such as "coastiness," "bush sickness," "enzoötic marasmus," "Nakururitis," etc. In this condition the animals become unthrifty, weak, emaciated, unproductive and, unless the condition is remedied, die.

Intravenous Medication[4]

By intravenous medication, we refer to the introduction of drugs, serums or blood, directly into the circulation of animals for the correction of disease conditions. The value of this means of medication is generally recognized by the veterinary profession and is used extensively in the treatment of disease by both the large and small animal practitioner. By means of intravenous medication it is possible to get much more rapid effects from the therapeutic agents administered, and as a result, many animals that formerly died are now being saved. Prussic acid poisoning is a very striking example of this. Where formerly practically all animals died after eating plants containing these poisons, it is now possible to restore these prostrated animals to normal health within a few minutes by the injection of neutralizing agents directly into the blood stream.

Perhaps the most common use for intravenous medication in large animals is in the treatment of parturient paresis in dairy cattle.

Blood transfusions are being more extensively used by the veterinary profession with each succeeding year. They are often indicated in cases of severe shock or accidental injury, and certain toxic conditions, such as sweet clover poisoning of cattle. When blood transfusions are to be used in valuable, purebred horses, blood typing is recommended, although it is usually unnecessary. In cattle, so far as is known, different blood types do not occur.

In the treatment of many infectious diseases, the intravenous administration of antisera gives more rapid and beneficial results than other types of administration.

For the administration of certain anesthetics, the intravenous route is gaining in favor. The ease and safety with which these anesthetics can be administered in this manner, have resulted in better and more humane surgery.

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[4] Abstract of a paper by W. R. Krill, D. V. M., Department of Veterinary Surgery, Ohio State University, Columbus, Ohio.

Susceptibility of Guinea Pigs to Equine Encephalomyelitis Virus[7]

Various sporadic, enzoötic and epizoötic nervous diseases are known to occur among horses, but since the widespread occurrence of the specific infectious disease designated equine encephalomyelitis, there is a tendency to classify all nervous diseases manifested by general symptoms as encephalomyelitis. This practice if continued will lead to error in evaluating the effectiveness of vaccination and treatment.

A varying incidence of the occurrence of encephalomyelitis in herds of susceptible horses has been noted by all investigators. Undoubtedly several factors have to do with this, such as the virulence of the virus, the dose inoculated, the site of inoculation and the resistance of the host.

Meyer and other investigators have shown that all laboratory animals can be experimentally infected by inoculating them intracerebrally with infective material. When other sites for inoculation are used, the results may vary greatly. This experiment was undertaken to determine the percentage of reliability of the various methods of inoculation of infective material from cases of encephalomyelitis into experimental animals. The objects were first, to provide a standard to evaluate results of vaccination or treatment and secondly, to establish a standard whereby one can more accurately evaluate negative results following inoculation of suspected material.

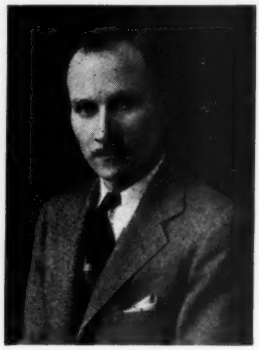
Guinea pigs were selected as the experimental animals. Inoculations of suspensions of brain in saline solution were made intracerebrally, intralingually, subcutaneously, intraperitoneally and intracutaneously into the foot pad of the left hind foot and the tip of the nose. Similar suspensions and doses were inoculated into all animals. This was repeated four or more times for each group. All of the animals receiving intracerebral inoculations succumbed. Results varied when other methods of inoculation were used.

[7] Abstract of a paper by Carl F. Schlotthauer, D. V. M., Division of Experimental Medicine, The Mayo Foundation, Rochester, Minnesota.

Composition of the Alveolar Air of Domestic Animals[5]

A knowledge of the composition of the alveolar air is essential in the explanation of the principles of respiration.

The alveolar air sample was collected and was transferred to the gas analysis apparatus. The volume percentages of oxygen, carbon dioxide, and nitrogen were determined. The carbon dioxide was absorbed



Carl F. Schlotthauer

from the unknown sample with 10% sodium hydroxide, the oxygen was absorbed with alkaline pyrogallol and the nitrogen and inert gas volume was determined by difference. With these volume percentages the partial pressures of the oxygen and carbon dioxide were determined. The partial pressure of the gases in the alveolar air determines the rate of gaseous exchange between the organism and its environment.

The results obtained in the horse were very similar to those obtained by N. Zuntz and his coworkers. They are also similar to those obtained in man by J. S. Haldane. The results for other species investigated compare favorably with the above. Therefore it may be concluded that the same principles of gaseous exchange that apply to man are also applicable in those species investigated.

A summary of the average composition and partial pressures as were determined in this investigation was given in tabular form.

[5] Abstract of a paper by George T. Edds, A. & M. College of Texas, College Station, Texas.

Variable Factors in the Measurement of Red Cell Sedimentation[6]

The phenomenon of settling out of blood cells from their fluid medium has been known since the time of Galen in the second century. However, apparently little clinical significance has been attached to it until within the past quarter-century. During this time, sedimentation, as the phenomenon is now referred to, has been studied in a great variety of disease conditions, with a resulting general consensus of opinion that it is a delicate non-specific measure of alterations in the fine physico-chemical balance of the blood, these alterations the result of destructive disease. With the wave of enthusiasm accompanying the "rediscovery" of the phenomenon, there has come into use a multiplicity of techniques, some basically sound and some not. This has brought only confusion in the measurement of this phenomenon. Theoretically there are many factors, variations of which may introduce errors, great or small, in the determination and interpretation of sedimentation. Some of these are:

1. Inadequate history of the source of the blood sample may lead to incorrect interpretation of results, especially failure to take into consideration such physiological states as pregnancy and estrum.

2. Failure to adopt a uniform procedure of obtaining blood samples may magnify errors produced by manipulation or venous stasis, or the like.

3. Too much or too little anticoagulant may exaggerate the undesirable effects already present in this variable but necessary requirement for determining sedimentation.

4. Insufficient agitation of the sample prior to starting the measurement of the phenomenon will fail to adequately disperse the solid elements and will thus render comparison of curves of sedimentation incorrect.

5. Capillary influence in small bore tubes is said to decrease the accuracy of determining sedimentation. It is suggested that shorter tubes allow less accuracy of measurement than longer ones. Deviations from perpendicularity of the tube and variations in the uniformity of bore introduce variables rendering comparison of samples inaccurate.

R. E. Nichols

6. Delay in determining sedimentation following bleeding apparently introduces a variable retarding influence on sedimentation.

7. Extremes in the external temperature at which the test is conducted alter the reaction.

8. Theoretically variations in the number of solid elements must necessarily induce variations in the curve of sedimentation, but it is dubious whether correction of samples to a chosen number of elements can be accomplished without altering other important physico-chemical factors influencing sedimentation.

9. Continuous examination (photographically) indicates that the rate of descent of the line of demarcation between cells and plasma, especially in the horse, is represented by a sigmoid or S-shaped curve. To answer the questions as to whether there is any deviation from the normal variation of the rate of descent of the line of demarcation between cells and plasma, and what kind and how much deviation, one must compare entire curves of sedimentation and not single readings or indexes. A single reading of the position of the line of demarcation between cells and plasma or any other procedure which does not at least attempt to portray the phenomenon as a continuous procedure, in all probability fails to measure the actual phenomenon of sedimentation.

[6] Abstract of a paper by R. E. Nichols, D. V. M., College of Veterinary Medicine, Ohio State University, Columbus, Ohio.

Equine Encephalomyelitis[8]

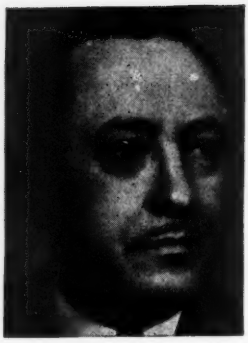
In 1935, there were 23,512 cases of equine encephalomyelitis; in 1936, approximately 4,000 cases were reported; in 1937, 170,000 cases were reported, and, in 1938, the outbreak assumed its largest proportions, 184,662 cases being reported to the Bureau. During 1938, 39 states were involved; eleven in the East and the other 28 west of the Appalachians. The general incidence for the 39 states involved was 23.5 cases per 1,000 animals, with a mortality of 21.4%. In the 11 eastern states, the incidence was 3.9 cases per 1,000 animals with a mortality of 90.2%. The incidence in the 28 states west of the Appalachians was 24.8 cases per 1,000 animals, with a mortality of 19.8%.

Results from the use of chick vaccine continue favorable both in experimental animals and in the field. Immunity tests recently made show that even after 12 months, vaccinated animals possess a high degree of immunity.

The veterinary corps of the army has vaccinated all the horses in the army, National Guard and R. O. T. C. units with vaccine prepared by the veterinary laboratory division of the Army Veterinary School. About 35,000 horses and mules were vaccinated between May 1st and June 15th with no unfavorable results or disturbing reactions.

Every effort has been made by the Bureau, with the full coöperation of the various firms, to see that only highly potent vaccine is marketed. In this plan of supervision, the Bureau has tested over 300 serial lots of vaccines prepared by the various commercial firms, representing about 20 million cubic centimeters of vaccine. In carrying out these tests, the Bureau has used over 5,000 guinea pigs.

Some unfavorable reactions from the vaccine have been reported from its field use. Investigation led to the conclusion that the vaccine was not to blame. It is believed untoward sequels to vaccination so far reported have been due to (1) faulty technique (contamination) in administering it, (2) pressure due to making the injection too rapidly and to depositing the vaccine all at one place, and (3) sensitivity to chick protein or to protein in general.



Harry W. Schoening

[8] Abstract of a paper by H. W. Schoening, V. M. D., Chief, Pathological Division, Bureau of Animal Industry, U. S. Department of Agriculture, Washington, D. C.

Pneumonia in Horses[30]

The clinical evidence of pneumonia in the horse tends to indicate that pneumonia is a secondary condition, and that it is rarely, if ever, a primary disease.

The primary agent in pneumonia of the horse appears to be a filterable virus which affects the upper respiratory and adjacent organs, and possibly the digestive organs. The virus infection lowers the natural resistance of the animal body to various types of secondary invaders. The streptococci are predominant among the secondary invaders and constitute the immediate causative factor in pneumonia.

There appears to be no specific treatment for pneumonia in the horse. Some of the newer drugs such as sulfapyridine, which have given such amazing results in human medicine, have in my experience, little if any value over some of the older, time-tried preparations in the treatment of pneumonia in the horse. Preventive measures, segregation and elimination of exposure to virus diseases and streptococcic infections, should receive first consideration. Light, cleanliness, ventilation, nursing and feed play a large part in the treatment and prognosis. The skill, ingenuity and observation of the practitioner is taxed to render aid and supportive treatment to keep up the metabolism and the natural and normal functioning of the organs of the body, especially of the heart.

[30] Abstract of a paper by D. L. Proctor, D. V. M., Lexington, Kentucky.

Mule Practice in the Sugar Cane Belt[33]

The mules used on the sugar plantations in Louisiana vary in weight from 1,000 pounds to 1,450 pounds. Those averaging 1,300 pounds predominate. They are subject to such equine ailments as influenza, forage poisoning, swamp fever, periodic ophthalmia, colic, septicemia, encephalomyelitis and anthrax.

Most of our cases of influenza are in the first stages of the disease. For mild cases, 5 to 8cc doses of equine influenza bacterin (mixed) are given at two-day intervals. In the more severe cases, 4.5gm of neosalvarsan in 20cc of distilled water is injected intravenously, repeating this dose in two days if necessary. Pneumonia appears infrequently, usually as a complication of influenza, and the termination is usually fatal.

Forage poisoning appears only occasionally, as the owners have learned the danger of moldy feeds. Swamp fever and periodic ophthalmia occur to a limited extent.

Colic is present in all its forms. For this, we give first a hypodermic of pilocarpine to stimulate the intestinal secretions and enhance the fluidity of the bowel contents. If there is any evidence of gastric flatulence, the stomach tube is passed at once, and one ounce of formalin in three to five gallons of water is pumped into the stomach and siphoned out. If there are borborygmi, we follow in 15 to 30 minutes with a hypodermic injection of arecoline hydrobromide. Most of these cases, after passing a quantity of flatus and excrement, make a good recovery. Cases in which the animal becomes ill while at rest, with a history showing nothing out of the ordinary, and with few of the usual symptoms present, usually develop into bowel stasis with progressive strangulation, and almost invariably terminate fatally. Autopsy reveals a section of the small intestine from three to 20 feet long in a gangrenous condition. The majority of these cases appear to be due to volvulus or intussusception.

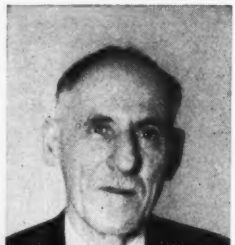
Wounds from nails, barbed wire, fork tines, soy bean and cane stubble often lead to streptococcic septicemia. These cases usually respond to the intravenous injection of 100 to 125cc of a 1% solution (aqueous) of mercurochrome at 48-hour intervals. Sometimes the treatment is repeated in 24 hours if the temperature is above 105°F.

Anthrax of the most virulent type is an annual visitor in Louisiana during the late summer months. For immunization against this disease we use the intradermal method, first injecting ½cc of a No. 3 spore vaccine in the side of the neck and following in 14 days or later with a like quantity of a No. 4 vaccine in the opposite side of the neck. This method of immunization against anthrax has proved very reliable. We treat a few cases of anthrax each year, using intravenous medication exclusively. Depending on the clinical symptoms and temperature, we use either anthrax serum, 100cc to 300cc, or 125cc of a 1:2500 solution of metaphen, occasionally alternating the two treatments.

A method worked out to prevent some of the principal ailments affecting mules on the plantations gives good results where the animals are housed, fed, and worked under the proper conditions. We begin in the spring by thoroughly fasting the animals for 30 to 36 hours and administering a carbon disulphide capsule for bots, giving five drams each to the medium size mules and six drams to the larger. At the same time we administer the first intradermal vaccine for the prevention of anthrax. Since the bot capsule has finished its work in two to three hours, and the absorption of the vaccine has hardly begun in 24, no clash occurs, and two very important preventive measures are handled at one visit. Another reason for administering the vaccine at this time is for its tonic effect. It has been our experience that the animals, following this treatment, shed their winter coats much more quickly and take on their true color much earlier. After the expiration of two weeks we again visit the plantation, the mules having been fasted again for 30 to 36 hours. This time we administer a No. 10 capsule of carbon

[33] Abstract of a paper by J. Arthur Goodwin, D. V. S., New Iberia, Louisiana.

tetrachloride for the expulsion of worms, and give the final dose of anthrax vaccine —again combining two important prophylactic measures in one visit, thus reducing the cost of the service. These treatments are given in the spring at a time when the field work is heaviest, and even those animals treated for the first time improve in condition very perceptibly, and remain in excellent health and condition the year round. After the crops are "laid by" (between June 15 and July 1), we do the dental surgery.



William E. Cotton

To keep records of these five separate prophylactic measures, we use a portable card index, with 3x5 inch cards upon which the name, address, date of service, kind of service, number of animals, etc., are entered. Since this is group treatment exclusively, records are kept for groups rather than for individuals.

The average life of mules on sugar cane farms is approximately nine years under the "hit-or-miss" plan; it is estimated that this "combined prophylaxis" adds from five to 10 years to their usefulness, which is quite a saving considering that the price of these mules ranges from $250 to $325 when purchased.

Blackstrap molasses, a by-product of the manufacture of cane sugar, enters largely into the daily ration fed the mules on the cane farms, and we have often been asked whether this product has any deleterious action upon the teeth. Apparently it does not; caries, alveolar abscesses and other irregularities occur just as often in mules not fed molasses, and frequently mules well over 20 years of age which have been fed molasses all their lives, have perfect teeth.

The Control of Bang's Disease in Dairy Herds[13]

The discovery of new facts concerning Bang's disease is of infrequent occurrence.

The control of Bang's disease in dairy herds usually differs from its control in beef herds and is generally more difficult.

The Bang's disease problem in herds supplying fluid milk for cities has become acute in many localities because of the fear of contracting undulant fever that is entertained by the public. This apprehension is not justified. During 1937 but 93 deaths of persons were reported to the U. S. Public Health Service. This disease is therefore a minor, a very minor health problem.

During the 59 months that the federal-state Bang's disease control and eradication program has been in operation, July 1, 1934 to May 31, 1939, 32,993,599 agglutination tests have been made throughout the United States in 2,653,730 herds; of these herds 509,361 were found to be infected and of the 13,723,858 animals in the infected herds 1,764,878 reacted to the agglutination test. In the same period 1,157,532 agglutination tests have been made in 53,972 herds in Alabama. Infection was found in 9,750 of these herds, which contained 679,091 cattle of which 44,673 reacted to the agglutination test.

The agglutination test is the best practical means we have of detecting infection in the field. A more efficient and less expensive method of handling the disease is urgently needed, but until a better plan than the test-and-slaughter method is developed it is our duty to make the best use we can of the knowledge and tools available to us.

Somehow the idea has become fairly widespread that agglutination tests made during a period extending for several weeks before and after the end of the gestation period are not accurate. This belief appears to be without foundation. At the Bethesda Experiment Station and at Beltsville when the writer was there, it was the

[13] Abstract of a paper by W. E. Cotton, D. V. M., Professor of Preventive Veterinary Medicine, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Alabama.

practice to test all of the cattle in vaccination experiments at short intervals throughout the gestation period and beyond, and in none of this work was there any indication that the state of pregnancy had any influence on the accuracy of the agglutination test. Moreover, experience in the field in connection with the federal-state Bang's disease program seems to support these observations.

There has been a good deal of controversy over the fate of suspects, that is, animals reacting in titer too low to be called reactors. Some contend that such animals should be removed along with reactors as the most of them will finally react in high titer. Experience gained in the federal-state Bang's disease program seems to indicate that on subsequent tests a considerable number of these animals become negative, some become positive and the remainder continue to be suspects. Information supplied by Dr. R. E. Jackson, B. A. I. Inspector in Charge of Alabama, shows that of 368 suspects retested during the two-year period, June, 1937 to May, 1939, inclusive, 253 (69%) were found to be negative, 87 (24%) positive, and 28 (7%) continued to be suspects.

It should be borne in mind that cattle reacting in titers of 1 to 100 and 1 to 200 are not all actually infected. In fact, *Brucella abortus* cannot often be demonstrated in the milk of cows that continue to react with a titer not above 1 to 100, and even those reacting in titer of 1 to 200 have by no means all proved to be infected. Of animals continuing to react in titer of 1 to 400 or above for considerable time, a very large proportion are likely to have infected udders. Since a small percentage of the cows that react at 1 to 100 and no higher and a considerable number of those that react at 1 to 200 are infected, it follows that in any program of control or eradication based on the agglutination test, that a good many animals that are not actually infected must be removed in order to get all of those that are infected.

In regard to vaccination, it seems to be pretty generally believed that the use of a living vaccine on adult animals is, at present at least, inadvisable, because of the danger of implanting the vaccine organism in the udder and its elimination in the milk if virulent cultures are used, and its interference with any method of control or eradication based on the agglutination test if living *Br. abortus* of any degree of virulence is used.

As has been explained by the writer on a number of occasions, these objectionable features can be largely overcome by confining vaccination to calves from four to eight months of age and using a strain of *Br. abortus* of low virulence in preparing the vaccine. As has frequently been pointed out, vaccination of this kind has yielded encouraging results at the Experiment Station of the Bureau of Animal Industry and is now being tried under the direction of the Bureau on about 10,000 animals in herds having an incidence of infection of 15% or more.

Incubating Hen's Egg As a Culture Medium for Brucella Abortus[12]

This investigation was undertaken to ascertain: first, whether the repeated egg to egg transfer of *Brucella abortus* will alter the virulence or the cultural characteristics or the antigenic properties of the organism, and second, if the incubating hen can be used to type different strains of *Br. abortus*.

All eggs were inoculated through the natural air sac on the tenth day of incubation. Strain No. 19 and a recently isolated virulent strain (Strain C. G.) were used throughout the experiment. At the start of the experiment, a series of guinea pigs and eggs were inoculated with graduated doses of Strain 19 and another series with Strain C. G. The lesions produced were noted for comparison with a similar set of inoculations at the end of the experiment.

Two eggs inoculated with approximately 130,000 organisms of Strain 19 and two inoculated with approximately the same

[12] Abstract of a paper by H. J. Metzger, D. V. M., Agricultural Experiment Station, New Brunswick, N. J.

number of Strain C. G. were selected for the inoculation of another set of eggs. The inoculum was prepared by grinding an asceptically removed liver and washing the sand with 20cc of sterile saline. Two tenths of a cubic centimeter of a 1:8 dilution of the 20cc suspension from each liver was inoculated into each of two eggs.

With each transfer the egg living the longer period was selected for the inoculation of two more eggs. The number of organisms inoculated at each transfer were counted by plating on tryptose agar, 0.1cc of a 1:10,000 dilution of 20cc suspension.

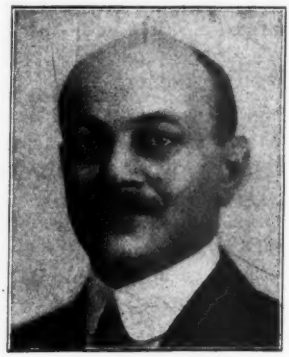
The chorio-allantoic membrane, liver, spleen and brain of all embryos were cultured on potato agar. All embryos living on the seventeenth day of incubation were killed and cultured. All cultures were checked for contamination by microscopic examination. A record was kept of all lesions observed in the embryos. One culture from each egg was incubated aerobically.

Suspensions of Strain 19 and of Strain C. G. from the eleventh transfer and from the original cultures of these two strains were inoculated into guinea pigs. The pigs were killed at six weeks and the lesions produced, the blood titer and the cultural characteristics of the organisms all indicated that neither Strain 19 nor Strain C. G. were changed in any way by eleven egg to egg transfers. Strain 19 continued to grow aerobically throughout the experiment. Antigen produced from these two strains obtained from the eleventh transfer checked exactly with our stock antigen.

The possibility of using the incubating egg for the purpose of typing different strains of *Br. abortus* was investigated by inoculating the series of eggs, one with different doses of Strain 19 and the other with different doses of Strain C. G. The dosages varied from 150 to 13,000,000. The length of time the embryo lived after inoculation and the lesions produced were recorded. A careful study of these records indicates that there is no significant difference between the effects produced by Strain 19 and those produced by the more virulent Strain C. G.

The Behavior of Brucella Vaccine in Various Excipients in Animal Inoculations[14]

While vaccination against Brucellosis in cattle has been practiced for many years



Adolph Eichhorn

and numerous publications are available on the results both with living and killed vaccines, data pertaining to the mechanism of absorption of the vaccines are very limited.

Studies were undertaken primarily for the purpose of determining the length of time required for the absorption of the living vaccine from the injected area, also to incorporate cultures in various excipients in order to establish whether it might not be possible to prolong the absorption from the point of injection, in order to enhance the immunizing action.

The experiments were carried out on cattle and rabbits. Bureau of Animal Industry *Br. abortus* strain 19 was used in all instances. Aside from the usual saline suspension, oil of vaseline, oil of sweet almond, cholesterol, saponin and lanolin were used as excipients. The studies included the behavior of the Brucella organisms in these excipients *in vitro* as well as *in vivo*.

It was found that, unlike other organisms, the Brucella suspended in salt solution, remained viable at the point of inoculation for at least 60 days. While at that period the number of colonies obtained from the inoculum were only few, on the

[14] Abstract of a paper by A. Eichhorn, D. V. S., C. K. Mingle and F. M. Murdock, Animal Disease Station, Bureau of Animal Industry, U. S. Department of Agriculture, Beltsville, Md.

other hand when the organisms were incorporated into cholesterol and other excipients, large numbers of colonies were obtained for longer than 60 days following the subcutaneous injections.

The studies also included the examination of various regional lymph glands and other tissues for the presence of Brucella organisms in the vaccinated animal. Throughout the experiments, the agglutination titer was studied in the various animals.

It is proposed to continue the experiments for studies of the relationship of the prolonged absorption of the vaccine to the degree and duration of the immunity, as well as the safety of the procedure for adaptation to calfhood vaccination.

The Gastrointestinal Worm Parasitisms of Cattle[10]

This is a study of the results obtained from more than 8500 examinations of bovine fecal samples. The sampling was made at monthly intervals from February 1937 to May 1937 on a group of 66 experimental animals kept at the Veterinary Experiment Station, Ithaca, New York. These animals were purchased as dairy heifer calves ranging in age from six to eight months, from a large number of dairy farms in Central New York State. During the 28 months of testing they were provided with good pasturage during the warm months and with a good grain and hay diet during the winter and were comfortably housed. Six of the animals were removed from the herd during the period. The samples were obtained manually from the rectum and a slight modification of the Stoll technique employed for examination. Five different counts were made from each sample examined and the results in terms of the number of worm ova per gram of feces were recorded for the Strongylid, Nematodirus, Trichuris, and Moniezia spp.

During the period of investigation most of the cows completed one gestation period and many of them had calves at the side. No outbreaks of acute infections occurred during the period and for the past year and a half no clinical evidence of internal parasitism has been observed.

In general, the data indicate that there was a period of about a year during which marked fluctuations in the worm burdens occurred. During the remainder of the period the counts of worm ova became stabilized at a low level in most of the animals.

[10] Abstract of a paper by Donald W. Baker, D. V. M., Experiment Station, New York State Veterinary College, Cornell University, Ithaca, New York.

The Sensitization of Cattle to Mammalian Tuberculin[11]

There is experimental evidence substantially proving that sensitization by the avian tubercle bacillus is responsible for many nonspecific reactions of cattle to mammalian tuberculin. Avian tubercle bacilli have been isolated from the lymph nodes of two naturally infected cows of an experimental herd composed of 15 tuberculin-reacting cattle from various herds with no history of bovine tuberculosis, and from one other tuberculin-reacting cow. After slaughter no gross lesions were found in any of these animals. Also, avian tubercle bacilli were isolated from the lymph nodes of three of 21 tuberculin-reacting cattle in which lesions were observed during routine inspection at packing plants. In none of these cases were bovine tubercle bacilli found. Two pure cultures of virulent avian tubercle bacilli and three mixed cultures of virulent avian tubercle bacilli and diphtheroids, inoculated subcutaneously, have previously been found to sensitize cattle to mammalian tuberculin. This paper describes the identification of an avirulent strain of avian tubercle bacillus isolated from a tuberculin-reacting cow. This culture when inoculated into calves sensitized them to mammalian tuberculin although it was virtually non-pathogenic for them.

The conclusion reached from this experiment is that even though bovine tuberculosis is being eradicated rapidly, cattle will continue to react to tuberculin until avian

[11] Abstract of a paper by Janet M. Carter, E. C. Hastings, and B. A. Beach, D. V. M., Departments of Agricultural Bacteriology and Veterinary Science, University of Wisconsin, Madison, Wisconsin.

tuberculosis is eradicated also. The ratio of these nonspecific reactors sensitized by the avian tubercle bacillus to specific reactors sensitized by mammalian tubercle bacilli may be expected to continue to increase until a more concerted effort is made to eradicate avian tuberculosis from chickens and hogs. This is an economic rather than a hygienic problem, since there is no evidence to show that avian tubercle bacilli are pathogenic for man under usual living conditions.

Induced Bloat in Dairy Cows[9]

Blood samples were taken under mineral oil, simultaneously, from the portal and jugular veins of sheep anesthetized with nembutal. These samples were analyzed for CO_2 content. Ligatures were applied to the esophagus near the cardia and to the abomasum near the omaso-abomasal orifice, thus confining the gas (CO_2) which was injected into the rumen to the first three compartments. After the gas had remained in the rumen for one minute, blood samples were again taken from the portal and jugular veins and gas analyses showed a marked rise in volumes % CO_2 (as much as 10 volumes %) in the portal blood, but no change in the jugular blood. CO_2 is absorbed quite rapidly from the first three compartments of the sheep's stomach and apparently dissipated in the expired air. The rate of absorption is directly proportional to the amount of pressure in these compartments. Later experiments showed that the rumen is the chief seat of absorption.

CO_2 pressure in the rumen causes a rise in blood pressure, an increase in heart rate and an increase in rate and depth of respiration in anesthetized sheep.

Carbon dioxide, methane, mixtures of CO_2 and CH_4, and air pressures have been established in cows with rumen fistulæ, at different measured pressures. CO_2 and CH_4 produce marked clinical symptoms, and very decided variations in respiration and rumen motility. Much higher air pressures have little effect on the animals, except to cause breathing to be much more shallow and to change rumen motility. Changes in rumen motility are quite similar in all types of gas. As the pressure increases, speed and amplitude of contractions are decreased. Rate and length of contractions increase as the pressure decreases. When air pressures in the rumen become quite high, the animals become very uneasy.

Absorption of CO_2 is much more rapid from a nearly empty rumen than from a full one.

This experiment was undertaken to simulate conditions existing in the rumen of cows affected with the "frothy" type of bloat, such as we have observed in postmortem examinations of cows that were bloated on irrigated ladino clover pastures. Gas and ingesta were completely mixed in the frothy mass, thus exposing most of the rumen wall to the contained gases.

Intravenous injections of sodium bicarbonate and sodium lactate are being made in an attempt to raise the CO_2 combining power of the blood. This method shows considerable promise. Ordinary treatments are practically useless in the "frothy" type of bloat that occurs on certain pastures.

Diseases Incident to Fattening of Lambs[15]

The vast area of the western range state comprises the breeding ground of the millions of lambs which are transported to the feed pens annually. The long distance on the trails to the loading points on the railways, and subsequent hours on board trains, especially in stormy weather, subject the lambs to infections through fatigue and exposure.

Hemorrhagic septicemia, nonspecific pneumonia, paratyphoid dysentery and sore mouth (contagious eccthyma) are the most common diseases causing heavy losses following such exposure. The practice of requiring a 12 to 24-hour fast period before weighing in at the receiving points on the railroads should be discouraged, and shrinkage figured on a percentage basis offered as a substitute. Lambs loaded immediately

[9] Abstract of a paper by R. W. Doughertv. D. V. M., Department of Veterinary Medicine, Oregon State College, Corvallis, Oregon.

[15] Abstract of a paper by N. J. Miller, M. D. C., Eaton, Colorado.

after such a fast period and then subjected to a 36-hour ride on the cars before being fed and watered, arrive in the feed pens in a weakened condition subject to almost any infectious condition. Long fast periods are recognized as being the definite predisposing cause of paratyphoid dysentery. Particularly in stormy weather, the unloading and feeding periods should not be extended beyond 24-hour intervals.

The sudden change from the wide open range with its natural grass feed to the close confinement of the feed pens and a more concentrated, heavy diet results in outbreaks of coccidial dysentery and loss from overeating. Lambs are particularly sensitive to indigestion and extreme care must be exercised especially in starting the lambs on their new diet, and this care must be continued throughout the whole feeding period until they are marketed.

Close confinement and errors in diet during the first three weeks in the feed pen are held to be the predisposing cause of coccidial dysentery.

"Overeating" does not, as the name might imply, mean a single or sudden engorgement, but is rather the product of the continued consumption of more concentrates than can be properly digested and assimilated. Overeating is a specific entity presenting characteristic symptoms and lesions, and probably causes more death loss in the feed lots in Colorado than all other troubles combined. An approximate ratio of the number of days on feed to the amount of concentrates fed must be maintained to control this condition.

"Lambing down" cornfields and pasturing of pea fields have been entirely abandoned in Colorado because of the heavy losses occurring under these conditions.

External parasites are of little consequence in the feed lot. Scab has practically been eliminated through federal regulations and ticks do little damage. Internal parasites cause very little death loss in the feed pens but the condemnation of livers in the slaughter houses because of distomiasis and the fringed tape worm cause an estimated economic loss of a quarter of a million dollars annually.

Preparturient Paresis in Ewes[16]

The obscure etiology renders experimental production of preparturient paresis in ewes unreliable. Since the disease is apparently metabolic in origin and is characterized by ketosis, lowered alkali reserve, and hypoglycemia (although a hyperglycemia is not uncommon) frequent blood analyses were made on 42 ewes divided into four groups and subjected to a variety of environmental and nutritional conditions. It was hoped that one of the groups, even if the disease was not produced clinically, would show a blood picture tending towards ketosis and a lowered alkali reserve as pregnancy advanced.

The first group was maintained on a dry pasture and fed sufficient well-cured alfalfa hay throughout the entire gestation period. The second group was maintained under similar conditions during the first three months of pregnancy and then moved to a small enclosure with concrete flooring and fed wheat straw and soy bean meal. The third group was maintained in a small enclosure and fed sufficient alfalfa hay during the entire gestation period. The investigations on these three groups were conducted between August first, when the ram was placed with the ewes, and January, when lambing finished. The fourth group was started in January and continued until June. They were maintained on pasture with alfalfa hay supplement for three months after the ram was introduced, after which they were moved to a small enclosure with concrete flooring and fed soy bean meal fortified with 50% cotton seed oil. No roughage was given.

The methods of analysis were as follows:

Sugar.—Oxalated blood was deproteinized by the zinc hydroxide method of Somogyi and sugar determined by the Benedict (1931) method. By this procedure glutathione and ascorbic acid were not included in the sugar values.

Alkali Reserve.—The plasma CO_2 capacity was determined by the Van Slyke method, using the manometric apparatus.

[16] Abstract of a paper by H. S. Cameron, D. V. M., and H. Goss, California Agricultural Experiment Station, Davis, California.

Ketone bodies, expressed as acetone, were determined by the method of Behre and Benedict. No analysis was made for ketone bodies unless the sugar or plasma CO_2 was abnormal.

Robert Graham

Results.—Of the 30 animals in the first three groups, four died from pneumonia, 17 had single lambs, eight had twins and one failed to conceive. No cases of preparturient paresis appeared in these groups nor was there any appreciable difference in the blood sugar level or alkali reserve in any group. In group 1 the blood sugar ranged from 17 to 51mg per 100cc of whole blood, with an average of 33.2mg; in group 2, from 22 to 44mg % with an average of 32.5mg; in group 3, from 18 to 40mg % with an average of 29.6 mg. The alkali reserve expressed as volume % CO_2 ranged in group 1 from 37 to 75mg with an average of 57.6mg; in group 2 from 45 to 67mg with an average of 57.7mg; in group 3 from 41 to 69 with an average of 54.8mg. Final results on the fourth group are not yet available.

In the field cases investigated an acidosis and ketosis were almost invariably present. Hypoglycemia was encountered much more frequently than hyperglycemia. A change in methods of handling and feeding in all cases stopped further losses.

Treatment by administering glucose intravenously was unsuccessful. It usually prolonged the life of the animal, but a relapse occurred when treatment was stopped. It lowered the ketone level of the blood. A quantitative analysis of the liver in one of the cases kept alive by glucose showed that the liver consisted of 71% of fat on a dry basis. The efficacy of treatment depends on the extent of liver damage.

Ketone Bodies, Sugar, and Calcium in the Blood of Nonpregnant Ewes[17]

Five nonpregnant ewes of good to choice quality, approximately 1½ years old and weighing an average of 115 pounds, were used for these studies.

All samples of blood were collected from the jugular vein. Either potassium oxalate or sodium fluoride was employed as an anticoagulant. The blood filtrates were prepared promptly from the oxalated samples and the analyses for sugar conducted shortly thereafter. Blood collected for calcium analysis was allowed to clot and the serum removed.

Effect of Exercise on Blood Sugar and Serum Calcium.—Moderate exercise, walking at a rapid pace for 15 minutes, produced no significant change in calcium and only a slight increase in sugar. Vigorous exercise, running at a gallop for 10 minutes, did not affect the serum calcium but resulted in a tremendous increase, an average of 85% over the pre-exercising level, in blood sugar.

Diurnal Fluctuation.—Samples of blood were collected at two- to three-hour intervals beginning at 8:30 a. m. and ending at 4:30 p. m. No pronounced fluctuations were encountered; the average for the calcium content remained nearly constant throughout the day, but the averages for total ketone bodies and sugar were slightly higher at the latter hour.

Fasting.—Water and salt were available throughout a fast of eight days. The results were: (1) A significant, though not a great increase in total ketone bodies; (2) a pronounced lowering, an average decrease of 30%, in the blood sugar content, which began on the second day of each fast, remained at a rather constant level until about the fifth day, and then gradually rose but not to the prefasting level; and (3) no change in the concentration of serum calcium.

Rations.—A series of experiments was

[17] Abstract of a paper by Jesse Sampson, D. V. M., L. E. Boley, D. V. M., and Robert Graham, D. V. M., Division of Animal Pathology and Hygiene, University of Illinois, Urbana, Illinois.

planned to determine the effects of certain feeds and combinations of feeds, fed at different levels for short periods. Results: (1) The concentration of total ketone bodies was not affected to any significant degree. The average for a total of 70 determinations was 1.56mg per 100cc of whole blood (expressed as acetone). (2) Blood sugar was decidedly affected. The average sugar content for the five ewes on a ration of two pounds of shelled corn and 2.5 pounds of alfalfa hay per ewe per day was 53.30mg %; on a ration of 3.5 pounds of alfalfa hay per ewe per day, 40.34mg %; on a ration of alfalfa hay fed ad libitum, 65.99mg %; and on a ration of two pounds shelled corn per ewe per day and alfalfa ad libitum, 68.98mg %. (3) The calcium content was not affected. The average for a total of 65 analyses was 12.03mg per 100cc of serum.

Intestinal Diseases of Swine[18]

Enteritis as a herd disease of swine is second only to hog cholera in its importance to the swine industry. The cardinal intestinal disease, necrotic enteritis, is a major problem in almost every neighborhood of the Corn Belt. It may be aptly compared in both its cause and lesions to the devastating outbreaks of human typhoid that once occurred in this country. Necrotic enteritis also leaves carriers which spread the disease to new premises, especially through community sales and newly purchased breeding stock.

Control of necrotic enteritis hinges on early diagnosis and full coöperation with local veterinarians, with regard to both sanitation and treatment.

Among the other intestinal diseases that play havoc with America's pig crop are (1) white diarrhea of suckling pigs, a condition that must be controlled by sanitation and proper feeding; (2) coccidial enteritis, a parasitic infection that is spreading rapidly in many sections; (3) hemorrhagic dysentery of feeder swine, and (4) the non-specific intestinal diseases due to toxic foods, salt poisoning, excessive or faulty feeding of irritant mineral mixtures, etc.

Inasmuch as enteritis occurs as a lesion in some of the specific, contagious diseases of swine—hog cholera, erysipelas, anthrax, etc.—the necessity for technical knowledge in the differential diagnosis of these troubles is self-evident. In fact, so confusing may be the various swine diseases of which enteritis is a part that confirmation of the diagnosis by a bacteriological laboratory is often necessary.

[18] Abstract of a paper by Frank M. Wilson, D. V. M., Mechanicsville, Iowa.

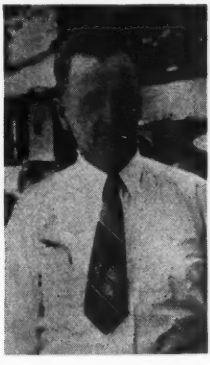
A Study of Death Losses in New-Born Pigs[19]

Death losses in new-born pigs present a serious problem for many hog raisers. A study of these losses has shown that they are frequently associated with the following gross lesions: Subcutaneous edema, particularly along the ventral side of the neck; and enlargement and increased friableness of the liver. Microscopically, the liver shows degenerative fatty infiltration; and the kidneys show parenchymatous degenerative changes which are mainly albuminous in nature.

The results of feeding experiments indicate that the ration the sow receives during the gestation period greatly influences the death rate in new-born pigs. When

[19] Abstract of a paper by L. P. Doyle, D. V. M., Indiana Agricultural Experiment Station, Lafayette, Indiana.

H. C. H. Kernkamp



L. P. Doyle

sows were fed a good quality of animal protein throughout the gestation period,[20] the average death loss of pigs during the first week after birth was 11%. When only grain and minerals were fed to the sows during the gestation period, the death loss of pigs was 44% during the first week after farrowing.

The Blood Picture in Hog Cholera[21]

Hemocytological studies on the blood of cholera-affected swine have shown that a definite leucopenia occurs in this disease. Decreases in the number of leucocytes equal to 30% and more over the pre-infection values have been observed in a great many cases. Significant quantitative cell differences are found to occur approximately 24 hours before any appreciable increase in the body temperature is noted, and about 72 hours before any other clinical manifestations develop. The differential leucocyte counts usually reveal a relative lymphocytosis, although this is seldom very marked. The cellular changes are generally most pronounced in cases of cholera that are not complicated by extensive inflammatory lesions or by parasites. A comparatively large number of swine have been used in these studies. With few exceptions all cases have been produced artificially with known hog cholera virus.

[20] *Veterinary Medicine*, 34:9, pp. 554-5.
[21] Abstract of a paper by H. C. H. Kernkamp, D. V. M., M. S., Division of Veterinary Medicine, University of Minnesota, St. Paul, Minn.

John D. Ray

Frank Thorp, Jr.

Aerobic Flora Occurring in Pneumonic Lungs of Swine[22]

The lungs from 204 swine submitted for autopsy were subjected to bacteriological and gross pathological examination. Thirty-eight of the 204 lungs were apparently normal and from one lung in this series a Streptococcus was isolated which corresponded to those of one group of Streptococci from pathological lungs. Forty-seven out of the total number showed various pneumonic lesions but no bacteria were isolated. From the remaining 119 pneumonic lungs, 153 isolations of bacteria were made. In 76.5% of these isolations, or 117 times microörganisms occurred alone or in combination with others of the same genus. Isolations of bacteria in combination with different microörganisms were 36 in number or 23.5% of the total. The bacteria isolated were grouped on the basis of morphological, cultural and physiological characteristics.

Gram-positive Streptococci were isolated 67 times, 64 cultures being of the *alpha* type and three of the *beta* type (Brown, 1919). Gram-positive Diplococci were isolated in ten instances. One gram-positive Staphylococcus was isolated which occurred in combination with a Streptococcus in one lung. Eleven cultures of gram-negative rods, for the most part inactive in the fermentable media, were placed in the genus Alcaligenes. A miscellaneous group of 20 gram-positive and gram-negative rods showing no uniformity in physiological characteristics were considered to be contaminants.

Most of the microörganisms were injected intraperitoneally into mice. The Streptococcus, Diplococcus and Staphylococcus cultures were relatively non-pathogenic for mice. Over half of the Pasteurellæ and about one-third the Alcaligenes and miscellaneous gram-positive and gram-negative rods were pathogenic for mice.

The Streptococci and Pasteurellæ were associated with the greatest variety of diseases. Hog cholera, lungworms, round-

[22] Abstract of a paper by Frank Thorp, Jr., D. V. M., Colorado Experiment Station, Fort Collins, Colo., and F. W. Tanner, University of Illinois, Urbana, Ill.

worms, infectious enteritis and pneumonia were the diseases with which the various microörganisms were most frequently associated.

The presence of colonies of moulds and actinomyces upon the two media used for primary isolation of microörganisms varied but little among the three groups of lungs, apparently normal and pneumonic, and therefore little if any significance can be attached to their presence. Yeasts or yeast-like fungi were not found in any of the primary isolations.

In this investigation pneumonia in swine occurred more often as a secondary than as primary condition. In frequency of occurrence the following genera and groups of microörganisms were isolated—Streptococci, Pasteurellæ, miscellaneous gram-positive and gram-negative rods (probably contaminants), Alcaligenes, Diplococci and one Staphylococcus.

Effects of Heat on Phenolized Hog-Cholera Virus[23]

For varying lengths of time, many temperature readings by a recording thermometer were taken during August and September, 1938, within the closed trunk, carrying compartments of several automobiles and within the closed seating portions of cars. Other readings have been taken in 1939.

With a view of determining more definitely the effects of heat on phenolized virus, particularly the thermal point or points at which it is rendered avirulent or non-viable, certain experiments were designed and conducted in an attempt to correlate them with field conditions under which simultaneous virus is ordinarily used by veterinarians in swine immunization.

Thirty experiments, involving approximately 425 pigs, were conducted. Each experiment was planned to include a sufficient number of pigs to justify the conclusions drawn.

Of 137 pigs inoculated with viruses heated at temperatures of 97 to 131°F., for one hour or longer, 133 pigs developed hog cholera.

Phenolized hog-cholera virus heated at 58.5°C. (137.3°F.) for one hour did not produce hog cholera in seven inoculated pigs; phenolized virus incubated at 36° to 37°C. (96.8°F. to 98.6°F.) for 168 hours produced hog cholera in only one of 17 inoculated pigs; phenolized virus heated at 36° to 37°C. for 144 hours produced hog cholera in seven of 30 inoculated pigs, divided into three test groups, in one of which eight pigs developed the disease through association with animals infected from the original inoculation; phenolized virus heated at 36° to 37°C. for 156 hours produced hog cholera in five of ten inoculated pigs.

On September 10, 1938, a recording thermometer placed in the rear of a closed trunk compartment of an automobile exposed to open sunlight recorded the following temperatures: 1:00 p. m., 119°F.; 2:00 p. m., 131°F.; 3:00 p. m., 136°F.; 4:00 p. m., 134°F.; 5:00 p. m., 122°F.; and 6:00 p. m., 103°F.

Phenolized virus heated in a serum pasteurizer on a temperature curve identical with the above failed to produce hog cholera in any of nine susceptible pigs.

The atmospheric temperatures recorded by the United States weather bureau for the same hours were: 92°, 94°, 95°, 88°, 87°, and 85°F., respectively.

It is, therefore, evident that hog-cholera virus may be rendered avirulent when transported in the closed, carrying compartment of automobiles for several hours where the normal atmospheric temperatures range from 92° to 95° to 85°.

The temperatures within the closed compartments of automobiles were found to vary widely, depending on the position of the car with respect to the direction of the sun rays, and other factors. The widest spread noted between atmospheric temperatures and car temperatures was 46°F.

Referring again to the data of temperatures in closed carrying compartments of automobiles, one notes that a high of 60°C. (140°F.) was recorded when the at-

[23] Abstract of a paper by J. D. Ray, D. V. M., and G. E. Whipple, D. V. M., Omaha, Nebraska.

mospheric temperature was 96°F. Since it has been reported by Munce and Hoffman and others that temperatures above 59°C. applied to anti-hog cholera serum as commercially prepared under license resulted in the development of shock-producing properties in such serum, it is believed that serum transported for a great length of time in closed automobile compartments exposed to direct sunlight, when atmospheric temperatures reach 96°F. or above, may likewise develop such shock-producing properties.

Control of Trichinosis in Hogs[37]

Surveys based upon human cadaver examinations show that there is a probable incidence of 17.25% of trichinosis in the human population of the entire country. Reported clinical cases, however, are few.

Surveys to determine the incidence and source of infection in hogs have shown that they may be placed in four groups: (1) those fed on cooked garbage 0.55%, (2) corn fed 1%, (3) fed raw garbage 5%, and (4) offal fed 10%.

The diagnosis of trichinosis is dependent upon sample testing of minute portions of muscle (diaphragm) either by direct examination, digestion or by intradermal testing of the living animal. Obviously these methods are too slow and laborious to be practical. Processing of known infected pork products to render them safe for human consumption is likewise impractical.

The control of trichinosis in hogs therefore reverts to the control of raw garbage and offal feeding.

It is recommended that garbage disposal and feeding be supervised by the state livestock sanitary and public health officials and that garbage be cooked before being fed to hogs. This would render pork from all hogs reasonably safe for human consumption cooked or undercooked. Further, it would aid materially the control of hog cholera.

[37] Abstract of a paper by L. E. Starr, D.V.M., Ph.D., Assistant Dean, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala.

Fowl Typhoid in Turkey Poults[31]

High mortality among week-old turkey poults produced in January by a large turkey grower was found to be caused by *Salmonella gallinarum*. The lesions in the affected poults were very similar to those produced by *Salmonella pullorum*. Hyperimmune serum produced in an adult turkey with the organism isolated from the poults gave complete agglutination in dilutions as high as 1 to 6,400 with this organism as well as with a stock culture of *S. gallinarum*, and in slightly lower dilutions with *S. pullorum* as antigen.

The adult turkeys on this farm had been routinely tested for pullorum disease with the following results: November 12, 1938, 8.7%; January 2, 1939, 3.3%; February 11, 1939, 1.4% positive reactors. About two weeks following the last test 2821 birds were tested in dilutions of 1 to 50, using both gallinarum and pullorum antigens. With the former 191, or nearly 7%, were positive, and with the latter, 170, or 6%.

Later poults of from six to 12 weeks of age became affected on this and several other farms; the mortality ranging from 20 to 50%.

In the outbreaks of fowl typhoid herein described it appears that *S. gallinarum* produced only a very mild form of disease in adult birds but that it was highly virulent for poults one to 12 weeks of age.

Duck Septicemia[36]

Antigenic strains of *P. avicida* bacterins have been found efficacious in controlling duck cholera on Long Island, New York.

Another disease of ducks apparently affecting the nervous system was found. The agent or agents responsible for this condition were not discovered. This disease did not appear to be associated with cholera, although it was found on some farms on which the ducks were dying of cholera. More often it was the only disease affecting the ducks on the farms.

[31] Abstract of a paper by E. P. Johnson, D. V. M., and Morris Pollard, Section of Animal Pathology, Virginia Agricultural Experiment Station, Blacksburg, Virginia.

[36] Abstract of a paper by Charles S. Gibbs, Lederle Laboratories, Inc., Pearl River, N. Y.

Effects of Intravenous Injection of Certain Salts on Intestinal Motility in the Dog[26]

This experiment included: (1) a study of the effects of different ions upon intestinal tonus and motility, and (2) a study of the influence of varying dilutions of sodium chloride on the intestine to indicate whether a detailed investigation was practical.

Two types of experimental procedure were followed: one recording movements of a balloon placed in the small intestine, the other recording the movements of a skin-covered, exteriorized loop of small intestine. In both, respiratory movements and blood pressure were recorded.

The effects upon intestinal musculature of sodium chloride in various percentage strength ranging from 2.5 to 12.5% were studied. A 5% solution of sodium citrate was used as well as a 5% solution of sodium bicarbonate and a 1% solution of calcium chloride. The effect of a 5% solution of calcium gluconate was studied. The salts of potassium included potassium chloride, 5% solution, and potassium bicarbonate in 5% strength. These salts were administered in various amounts by intravenous injection.

The following conclusions were made from these studies:

1. Solutions of sodium chloride stimulate intestinal musculature. The first injections of small amounts stimulate intestinal motility primarily. Subsequent injections of larger amounts stimulate intestinal tonus but suppress motility somewhat.

2. The quantity of sodium chloride injected rather than the percentage strength of the solution appears to be the significant factor. The response of the intestine varies in proportion to the amount of the salt injected.

3. Large amounts of hypertonic solutions of sodium chloride administered intravenously are relatively nontoxic to the circulatory and respiratory systems.

4. An increase in tonus is the first and most persistent response of the intestine to an intravenous injection of sodium citrate, intestinal motility being stimulated moderately. Bradycardia and slowing of respiration result from large injections of sodium citrate.

5. The sodium ion appears to be instrumental in producing a stimulation of intestinal musculature. Sodium chloride and sodium citrate both stimulate the intestine, but citric acid and a solution of hydrogen chloride gas do not affect it. Therefore, it appears that the citrate and chloride ions do not stimulate the intestine. The sodium ion appears to be the only remaining factor and therefore seems to be responsible for the stimulation of the intestinal musculature.

6. Lang's solution is a very effective agent in stimulating both intestinal tonus and motility. However, it is slightly toxic to the respiratory and circulatory systems of anesthetized dogs. Lang's solution appears to stimulate the intestinal musculature by virtue of the sodium ions present.

7. Sodium bicarbonate stimulates intestinal tonus and motility more effectively than any of the other solutions used, and produces little or no effect on the respiratory and circulatory systems.

8. Calcium chloride does not stimulate the intestine as much as the sodium salts, but the effects persist longer. Considerable danger of heart block accompanies the intravenous injection of calcium chloride.

9. Calcium gluconate stimulates the intestinal musculature more than sodium chloride. Boric acid, which is used to increase the solubility of calcium gluconate, apparently has no effect on the intestine.

10. The respiratory and circulatory systems are not altered significantly by the intravenous injection of calcium gluconate.

11. Potassium chloride and potassium bicarbonate appear to depress the intestinal musculature. Potassium chloride is exceedingly toxic to the heart.

[26] Abstract of a paper by L. M. Jones and E. A. Hewitt, D. V. M., Ph. D., Department of Veterinary Physiology and Pharmacology, Iowa State College, Ames, Iowa.

Studies in Canine Coccidiosis[25]

During the spring and summer of 1938, 320 dogs were examined for coccidial infection over a period of nine months. The fecal examinations were made according to Sheather's sugar flotation method as modified by Benbrook and Bourne.

Most of the animals were obtained from the surgical and medical wards; the remainder were boarders and strays, the latter brought in for euthanasia. Most of these dogs were examined at least every other day during their hospitalization period. Fourteen experimental animals were divided into two groups and kept in strict confinement for a period of eight weeks. Precautions were taken to prevent cross-infection between dogs and contamination of utensils during the time the fecal examinations were being made.

Of the animals checked, 79% harbored coccidial sporocysts or oöcysts, while 21% were negative.

Thirty-two per cent of the samples showed one to five sporocysts in each high power field, 23% showed five to 25 sporocysts per high power field, and 9% showed over 25 sporocysts and oöcysts per high power field.

The parasites were identified as belonging to the genus Isospora, the species being felis, rivolta and bigemina. No *Eimeria canis* were found.

Two experimental groups of animals composed of six and eight dogs were kept under strict surveillance for four and six weeks, respectively, and their feces were examined daily. Eleven of them were found to harbor coccidia; three were negative. None of the dogs showed any symptoms of coccidiosis. The organisms found were usually in the sporocystic stage.

Three animals received by mouth two doses of ¼gr arecoline hydrobromide to break the chronic stage and to obtain oöcysts in the feces. In each case the oöcysts were found in the second fecal passage eight to 10 hours after treatment. Two days later the feces again contained only sporocysts.

All the animals were sacrificed. Some were in the acute stage, due to the arecoline, while others were in the chronic stage. In each case the entire digestive tract was examined. All showed more or less injected areas in the mucosa, thickened walls due to an increase of connective tissue elements and congestion of the serosa, especially in the animals which had received arecoline a short time before autopsy.

The intestinal tract was transsected every three inches from the pyloris to the cecum and a small piece was removed, fixed and sectioned in paraffin for histological study. The remainder of the intestine was opened and some of the intestinal contents were saved for examination.

The intestine was washed thoroughly with tap water to remove all adhering material and then scraped. The scrapings were suspended in physiological saline solution, shaken well, centrifuged and the supernatant fluid discarded. After repeating this process three times the sediment was checked microscopically. In each case the villi, denuded of epithelium, showed coccidial organisms in various stages of development in the tunica propria. The lower part of the jejunum and the ileum contained the greatest number of parasites. None were found in the anterior part of the duodenum or the cecum.

It was found that the *Isospora bigemina* always matures fully in the subepithelial tissue of the villi, while the *I. rivolta* does so only occasionally. Single sporocysts were found in great numbers, as well as oöcysts just in the act of releasing their sporocysts. The histological study of stained paraffin sections corroborated the above findings.

Two dogs which after repeated fecal examinations were considered to be free from coccidia, were found to harbor a few of these parasites in the tunica propria of the intestinal villi. Photomicrographs of all important phases were taken.

[25] Abstract of a paper by F. X. Gassner, D. V. M., Colorado State College, Fort Collins, Colo.

Lesions of Gossypol Poisoning in Dogs Fed Cottonseed Meal[29]

Raw cottonseed meal was used in the ration as the source of protein at the rate of 27%. Dogs received as much of the feed as they would eat within a short time, approximately one-half pound twice daily. Normal gains were made even though the dogs apparently had to acquire a taste for the ration. After nine to 12 months on this ration symptoms of posterior incoördination of several days' duration, stupor or somnolence and lethargy observed a day or so prior to death occurred in these dogs. The dogs died suddenly and rather unexpectedly; they were in good flesh and coat at the time. No pathogenic organisms were isolated and the possibility of other infectious agents was largely precluded.

Gross changes were: Marked hydrothorax and moderate hydropericardium; edema, hemorrhage and small pneumonic areas at the ventral apical lobes of the lungs; the blood was increased in viscosity and dark in color; atony, hypertrophy, edema and areas of degeneration constituted the heart changes; moderate ascites, congestion, hyperplasia of the mesenteric lymph glands; enlargement, mottling, congestion and areas of hemorrhage of the liver; large suggilations and possible slight fibrosis of the spleen; the walls of the gastro-intestinal tract were edematous, thickened, numerous ulcerations or erosions of the fundic portion of the stomach and the first three feet of the intestine with a hemorrhagic exudate; surface of the kidneys was mottled, with areas of degeneration and infarction. There were no visible macroscopic changes of the nervous tissue.

Microscopic changes were: Chronic hemorrhagic hepatitis; chronic tubuloglomerulo nephritis; fatty degeneration and fibrous replacement of the myocardium; extensive edema, hemorrhage and small pneumonic areas in the lungs; edema, catarrhal-hemorrhagic exudate with degeneration of the surface epithelium and secreting tissue of the stomach and intestines; large patchy areas of demyelinization, axonic fragmentation and swelling of the peripheral nerves; swelling of several cells, irregularity and partial loss of Nissl granules constituted the central nervous changes.

Gossypol apparently has an endotheliotoxic action when fed to dogs in large amounts over a long period.

[29] Abstract of a paper by John L. West, Alabama Polytechnic Institute, Auburn, Alabama.

Streptococcic Infections in Dogs[24]

II. *Pathogenicity for Pups, Tonsillitis, Abscesses and "Acid Milk"*

No young pups were available for virulence tests until several months after the isolation of the streptococcus previously referred to as *Streptococcus canis*. Intraperitoneal and subcutaneous injections and feeding of broth cultures proved harmless except in the case of one pup, which also showed a rather heavy roundworm infestation. This streptococcus, therefore, either had lost its virulence or it is an opportunist which infects hosts rendered susceptible by some predisposing cause. Streptococci were found to be the cause of an outbreak of tonsillitis in nine pups and two older dogs, all in one kennel. Cultures made from abscesses (not post-vaccination abscesses as in an earlier investigation) have yielded streptococci in most cases. A moribund pup (a few days old), suffering from "acid milk," was chloroformed and immediately examined bacteriologically. *Escherichia communior* (hemolytic) was isolated in pure culture from the liver and spleen. This organism was also obtained from the lungs which, in addition, yielded a gram-positive coccus. Swabs made on the same day from the vagina of this pup's mother also yielded cultures of hemolytic *Esch. communior*, and, in addition, a diphtheroid and a Neisseria. Thus, it seems possible that more than one type of infection may pass under the name "acid milk."

[24] Abstract of a paper by H. J. Stafseth, D. V. M., Michigan State College, East Lansing, Michigan.

Modification of the Distemper Virus[34]

Distemper maintains a relatively virulent character for unrelated species. The virus isolated from natural cases of distemper in dogs or foxes usually produces fatal infections in ferrets. However, a study of many natural strains of distemper from various source animals shows marked variations in virulence of the disease for different species. It has been found in these investigations that the distemper virus is capable of modification by prolonged passage through a single species, so that its virulence is lessened for other susceptible but unrelated species. Serial passage of the distemper virus through ferrets or minks greatly reduces its virulence for foxes and dogs. The process of modification can be carried to such a degree that the virus, while extremely virulent for ferrets, produces a relatively harmless infection in foxes.

Twenty-five young foxes injected subcutaneously with a distemper virus obtained from an epizoötic in foxes, suffered a mortality of 84%, 21 of the animals succumbing to distemper. Two groups of 10 foxes each were injected with 4th and 10th ferret-generation virus, with losses of five and four animals, respectively, the total mortality being 45%. Three groups of 10 foxes each were injected with virus of 14, 15, and 18 serial passages, respectively. Eight animals died, a total mortality of 27%. Twenty-five pups were injected with 21st generation virus. Three, or 12%, of the animals died. Among 56 foxes injected with 27th generation virus, there were 5 losses, a mortality of 9%. The average mortality for the entire group was, therefore, 10%.

From this point on, larger groups of foxes were used for single tests. Inoculation of 50 foxes with 39th generation virus resulted in four deaths, a mortality of 8%. Fifty foxes were inoculated with 53rd generation virus and four deaths occurred, a mortality of 8%.

There was definite evidence that in the above tests natural distemper gained access to the groups and that some of the deaths, at least those produced by the 53rd generation virus, were due to accidental infections with the natural distemper virus. It was, therefore, decided to inject the virus into a large group of silver foxes in a unit free of distemper. Five hundred ranch foxes 4½ months old were injected with the 54th generation virus. The animals in this group exhibited no signs of illness after inoculation. Three of the foxes died during the next 90 days, but none of the three could be shown to have died of distemper. The total mortality for this group was 0.6% during the 90 days following injection of the live virus.

Simultaneously with the inoculation of the 500 foxes, an injection of the 54th generation ferret virus was made into 10 ferrets. All of the ferrets were killed *in extremis* on the eighth day, with infection typical of that caused by the highly-virulent passage virus in the homologous animal.

When tested with suitable controls, foxes that had previously been injected with modified virus exhibited a high degree of immunity to a distemper virus virulent for foxes.

During modification by ferret passage, the distemper virus goes through a continuous and gradual change in its pathogenic properties for an unrelated but susceptible species such as the fox or the dog. This change is more than a decrease in virulence. A distemper virus passed serially through ferrets for approximately 25 generations still produces a mortality of about 10% in young foxes, but the disease in foxes is no longer recognizable clinically as distemper. The animals show symptoms for only a few hours and die suddenly with necropsy findings that may easily be confused with those of fox encephalitis. The inclusion bodies present, however, are those of distemper. Upon further passages, the sudden deaths become fewer and finally the virus seems almost nonpathogenic for foxes. Nevertheless, a definite infection is produced by the virus.

[34] Abstract of a paper by R. G. Green, M. D., University of Minnesota, Minneapolis, Minnesota.

The modification of the distemper virus here described seems analogous to the change in the smallpox or variola virus as it is transformed by animal passage into the vaccinia or varioloid virus used in the immunization of human beings. It is suggested that the mild infection produced by the modified distemper virus be referred to as distemperoid and that the modified virus be called the distemperoid virus.

The Vaccination of Dogs with Modified Distemper Virus[35]

The modified virus used for the experimental vaccination of dogs had been passed serially through ferrets from 53 to 56 times. The virus in its final form was spleen collected from ferrets *in extremis*, and immediately frozen. Most of the vaccines for dogs were made with suspensions prepared directly from this frozen material. Other inoculums were made with desiccated virus. The dosage of the modified virus was in all cases 0.5cc of 0.5% spleen suspension. In no instance was any killed virus or protective serum used before or with the injection of live virus.

Isolated groups of dogs, ranging from one to 14 in number and totaling 114 animals, have now been injected with this modified virus without undue reaction, although the virus on frequent, and often simultaneous, tests showed extremely high virulence for ferrets. The dogs inoculated were mongrels, springer spaniels, and golden retrievers. Twenty-three were small puppies only three weeks old when injected; 76 were from four to 16 weeks of age; 10 were about eight months old; and five were adults. No difference in severity of general response had been noted in the different age groups. No severe reactions following the inoculations have yet been encountered. Most, but not all, of the puppies, however, have shown unmistakable signs of illness; partial loss of appetite, some conjunctivitis, looseness of the bowel, and listlessness for a day or two. Many of the dogs have shown a single reaction, about the 6th or 7th day, while others have shown a secondary reaction from the 12th to the 14th day.

A litter of eight golden retriever puppies injected with dehydrated virus on the 21st day after birth were intensively studied. Seven of the eight had a slight fever about the 7th day and failed to gain weight over a period of from two to four days. All the puppies were then free of fever and gained weight until about the 14th day, when all had a slight fever and gained no weight for several days. By the 3rd week after vaccination most of these puppies were expelling worms. They were dewormed on the 21st day and again on the 28th day. On the 24th day, six of the puppies developed from one to nine pustules on the abdomen, although at this time they were generally thrifty, gaining weight rapidly, and were very active. As a complication of the pustules, and probably partly as the result of injury, one of the puppies developed an abscess. With the exception of a temperature of 104.2°F. in this puppy previous to lancing of the abscess, the highest temperature noted was 103.6°F., and this was maintained for only one day. The mother of the dogs, 10 months old, showed a more severe reaction than any of the puppies. She had a slight fever on the 5th and 6th days and a temperature of 104.8°F. on the 11th day, which gradually dropped to 102.2°F. on the 16th day. She ate well throughout this period, except on the 11th day.

This group of puppies were tested for immunity to distemper 78 days after vaccination. They were given subcutaneously 0.5cc of 0.1% commercial, live, frozen, dog-spleen virus as a test of immunity. Temperatures of the puppies were taken for 18 days following the inoculation but no fever was evinced. One puppy did show a rise of two degrees in temperature on the 7th and 8th days as a result of an infected wound received in a fight with littermates, but its temperature was again normal on the 9th day. All puppies appeared entirely immune. A control ferret developed distemper after the usual period of incubation.

Another litter of three-week-old puppies

[35] Abstract of a paper by R. G. Green, M. D., University of Minnesota, Minneapolis, Minnesota, and F. S. Swale, D. V. M., Fromm Laboratories, Grafton, Wisconsin.

was inoculated with fresh modified virus. This group of 10 springer puppies did not show any marked or noticeable reaction other than the formation of pustules on the abdomens of three of the 10 on the 9th day. Temperatures were taken for a period of 18 days, during which time they remained normal. The mother of this litter had a slight fever of 1.2° on the 11th day, which was probably due to a foot injury. Her temperature was again normal on the 13th day.

No reactions were seen in the rest of the 23 puppies injected when three weeks old. The only development of pustules seen was in this group of very young dogs. In the animals in which pustules occurred, the general reaction was not more marked than in the others. During the time the puppies had pustules they were playful and ate well.

Although not a single severe reaction was seen among 109 puppies under fair to good conditions of care and feeding, four deaths resulted from virulent secondary invaders in a test conducted on 25 puppies in a highly infected sales kennel that handles low-priced dogs. This group represented a classical example of the influence of unsanitary conditions and malnutrition. It becomes obvious that the modified virus can be used as a vaccine only for dogs that are in fair health and nutrition and have some protection from sick and convalescent dogs carrying virulent microörganisms.

Practical use of a modified virus by Green and Carlson for the vaccination of the silver fox shows that such a virus is safe for use in that animal and results in an immunity sufficient to control completely distemper outbreaks on fox farms. The modification of the distemper virus by serial passage through ferrets offers an entirely new tool in the vaccination of dogs against distemper. The reduction in virulence effected by animal passage appears to be a dependable character of the distemper virus. From our results thus far, general vaccination of dogs by the judicious use of a single dose of modified distemper virus would seem to be a possible development.

Nervous Disturbances in Dogs[27] (Cinematograph)

This film and the explanatory statements gave the day by day appearance and record of two groups of dogs used in an experiment to determine the cause of fright disease.

Young dogs fed on a vitamin B_1-free diet of high carbohydrate content developed typical fright disease with almost clocklike regularity in five to eight days (depending upon individual tolerance) and recovered promptly when given massive daily doses of thiamin hydrochloride.[28]

The spectacular running fits and convulsions of fright disease were preceded by a longer or shorter period of progressively increasing nervous disturbance, during which the animal was restless, irritable, noisy, and showed an increasing lack of muscular control beginning in the hind quarters and accompanied by attacks of clonic opisthotonos and emprosthotonos. In severe, acute vitamin B_1 deficiency on an almost pure carbohydrate diet this whole syndrome might be experienced within 96 hours.

Vitamin B_1-free diets were prepared by autoclaving (after adjusting the pH) commercial dry dog foods for two hours at 15 pounds pressure. Various dry dog foods obtained on the open market were so nearly vitamin-free that the regular onset of fright disease in dogs fed upon them was not delayed more than 24 hours longer than was required to bring the same animals down upon a vitamin B_1-free diet. Other commercial dry foods contained an adequate quantity of B_1.

Reference was made in the explanation to a later experiment in which the fright disease syndrome was separated into nervous symptoms (irritability, restlessness, etc.), muscular incoördination and convulsions, and each group of symptoms produced separately from the other two. This result could be attained only in chronic cases where the dogs were supplied a maintenance ration of vitamin B_1 but deprived of other members of the vitamin B-complex.

[27] Abstract of a discussion by John W. Patton, D. V. M., M. S., East Lansing, Michigan.
[28] Veterinary Medicine, 34:6, pp. 372-81.

Blood Parasites of the Dog[32]

Small animal practitioners commonly make diagnoses of heart worm infection from the mere recognition of hematozoa in peripheral blood, and treat the animal accordingly. At least in some regions, however, only a small percentage of dogs whose blood is teeming with hematozoa are found on post-mortem examination to be positive for *Dirofilaria immitis*.

John W. Patton

While moderate heart worm infections appear to yield reasonably well to established therapeutic measures, similar medication has failed to affect the parasitism responsible for the other hematozoa. In order to avoid prolonged, expensive treatment in cases in which it is of no avail, early differential diagnosis is obviously desirable.

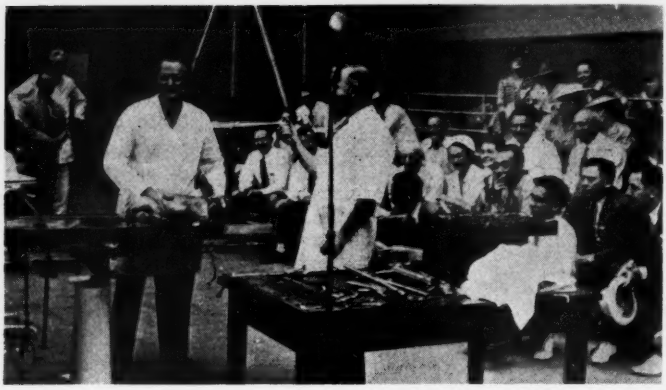
Differentiation is not possible on the basis of clinical examination. Post-mortem examination is, of course, conclusive in showing the presence or absence of heart worm infection, but is of no help in the problem of diagnosing the condition in the living dog. The authors have attempted to devise a method of diagnosis by allergic skin reactions or precipitin tests, but the close biological relationship of the parasites concerned have thus far prevented satisfactory differentiation by such methods. Detailed morphological examination of the hematozoa is conclusive, but is too complicated a procedure to be of practical use to the veterinarian. One difference between the two types of hematozoa is, however, recognizable microscopically with little difficulty: the *Microfilaria immitis* is of greater body width with a decidedly narrower, tapering cephalic extremity, while the unknown larva has a narrower body with a distinctly bulbous anterior end. A further recognizable point of variation is that the anterior somatic cells of *M. immitis* extend much nearer to the anterior extremity than do those of the unknown. The technique of preparing and staining the hematozoa for differentiation on these points is not difficult, and until a better method can be devised, this appears to be the simplest method of differentiation available.

The unknown microfilaria has not been definitely classified. However, in animals examined by the authors, 92% of those harboring the unknown hematozoa have been found on post-mortem examination to be infected with *Spirocerca sanguinolenta*, the so-called esophageal worm; while careful search has in no case disclosed any other adult parasite to which the hematozoa could possibly be ascribed. If, as seems possible, the unknown hematozoön is a definite larval stage of *S. sanguinolenta*, some careful rechecking is needed of the accepted life-cycle of this parasite; the more so in that several discrepancies have been noted in the life-cycle as described by Mönnig and others.

It has been suggested that the unclassified hematozoa represent a larval stage of the hook worm, but this possibility appears to be contraindicated both by outstanding morphological differences in the larvæ and by the exceedingly large number of hematozoa often present in the blood of a dog; the number of hook worm larvæ which might be expected to be present in the blood at any one time is relatively very low.

In view of the lack of facilities for extensive pet animal investigational work of a scope commensurate with its importance, the authors suggest that representatives of (1) dog organizations, (2) dog owners, (3) industries supplying canine products, and (4) small-animal practitioners, meet to take steps toward the coöperative erection and maintenance of a permanently endowed investigational institution of national scope, the work of which would cover all canine problems, both pathological and non-pathological.

[32] Abstract of a paper by R. L. Mundhenk, P. D., D. V. M., and J. E. Greene, D. V. M., M. S., Department of Anatomy and Histology, School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala.



J. Gilbert Horning, Houston, Texas, demonstrating the technique of esophagoscopy and bronchoscopy and expounding upon the many conditions in which these diagnostic or therapeutic measures are indicated.

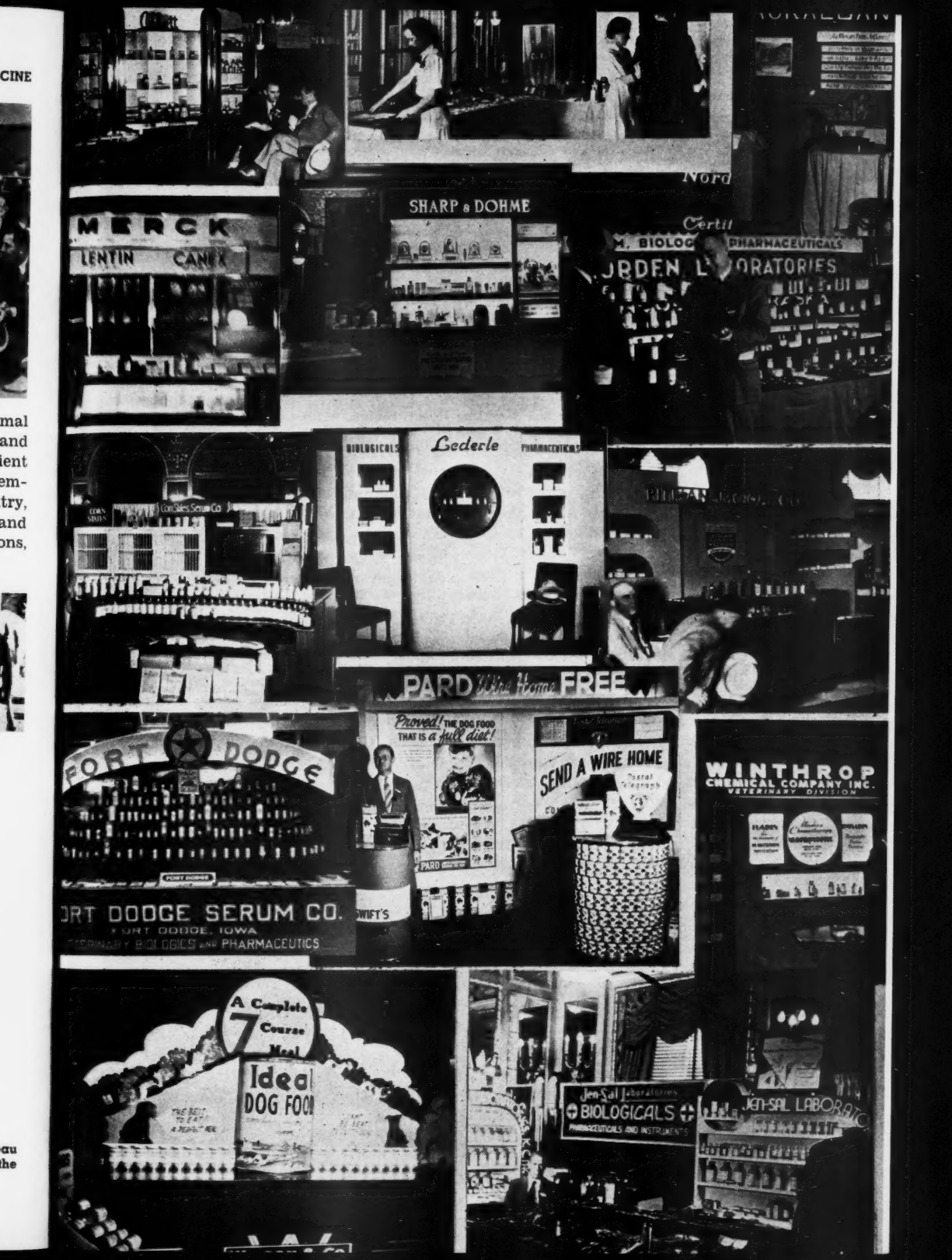
The Clinic

In amount and variety probably no clinic of the A. V. M. A. has exceeded that staged at Memphis. It included all domestic animals in ample numbers, with mules predominating. The horse and mule clinic was in charge of W. L. Gates; the cattle clinic, in charge of J. T. Alston; the small animal clinic, of E. B. Mount; the sheep, swine and poultry clinic, of C. P. Branigan. Sufficient material was available for continuous demonstrations during clinic day of one poultry, one swine, one cattle, two small animal and half-a-dozen horse and mule operations, simultaneously.

A Group of Clinic Subjects Awaiting Their Turn at the Hands of Noted Surgeons

Dean E. E. Wegner
received a round of applause for the careful technique and expeditious manner in which he operated upon a cryptorchid stallion in the clinic.

H. E. Moskey
constituted a sort of walking information bureau much sought for information as to provisions of the new federal Food, Drug and Cosmetic Act.


MERCK
LENTIN
CANEX
SHARP & DOHME
Lederle
BIOLOGICALS
PHARMACEUTICALS
PARD
FREE
Proved! THE DOG FOOD THAT IS a full diet!
SEND A WIRE HOME
FORT DODGE
ORT DODGE SERUM CO.
FORT DODGE, IOWA
WINTHROP
CHEMICAL COMPANY INC.
VETERINARY DIVISION
A Complete 7 Course Meal
Ideal
DOG FOOD
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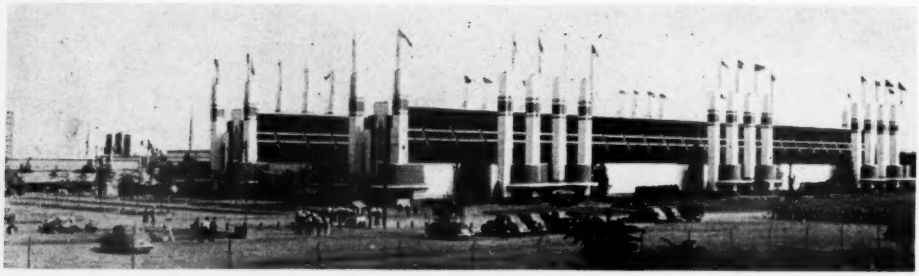
A. V. M. A. Day at World's Poultry Congress



HALL OF NATIONS AND STATES

In this building, one of those remaining from the Great Lakes' Exposition, the United States government, 29 states, the foreign possessions of the United States and 11 foreign countries had exhibits depicting the poultry industry of those regions and describing recent research which they have carried out.

The Seventh World's Poultry Congress and Exposition, held at Cleveland, Ohio, July 28-August 7, 1939, was a stupendous—a colossal affair. In the opening address on A. V. M. A. Day the chairman stated that attendance had exceeded the wildest guess of the most delirious promotor of the show. At that time, the paid admissions were approaching half a million and the Congress was not yet half over. It was stated that it could have been closed at the end of the third day and sufficient funds would have been available to pay every bill out of the receipts up until that time.

In advance of the Congress, more than 5,000 persons and firms had contributed from $10 to $3,750 each to the fund for organizing this, the first World's Poultry Congress to be held in the United States. In addition, more than $150,000 was raised from the sale of affiliated memberships at $1.00 each. In addition to these sums, were the large number of regular ($7.50), special ($3.00) and family ($10.00) memberships and the fees received from the more than 250 exhibitors.

Wednesday, August 2nd, was designated in the official program of the Congress and elsewhere as A. V. M. A. Day. In the afternoon of that day, the scientific program was exclusively veterinary and was attended, among others, by 230 veterinarians and their wives; an excellent attendance and a tribute to the energy and ingenuity of Cliff D. Carpenter, who had in charge the program and arrangements for the participation of the A. V. M. A. in the Poultry Congress.

The address of welcome was by Prof. James E. Rice of Ithaca, N. Y., general chairman of the congress, and the response by Dr. L. A. Merillat, executive-secretary of the A. V. M. A. Others contributing to the program were: John R. Mohler, Washington, D. C.; W. R. Hinshaw, Davis, Calif.; O. V. Brumley, Columbus, Ohio; C. A. Brandly, East Lansing, Mich.; Walter E. Brandner, Petaluma, Calif.; Frank C. Tucker, Claypool, Indiana, and Chas. B. Cain, Memphis, Tenn.

In his address of welcome, Prof. Rice reminded hearers that the poultry industry, which in this country ranks in size ahead of every other branch of animal industry except dairy, was less than half a century old, that 50 years ago there was no poultry industry anywhere, but only a great number of individuals that produced poultry as a side line.

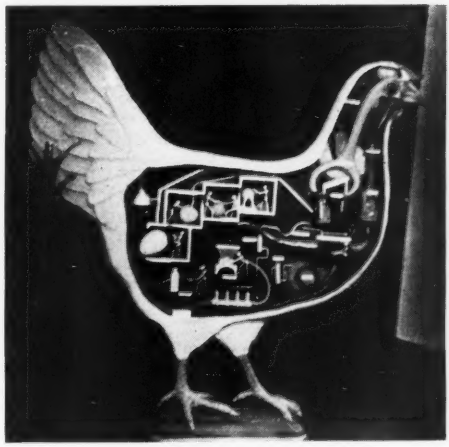
The World's Poultry Science Association was organized only 18 years ago by Prof. Rice. The first meeting of that association was held in 1921. A meeting with the Poultry Congress in conjunction with it, has been held every three years since. The Congress constitutes practically the whole show, but during the Congress the science association holds the meetings at which

papers are read detailing the advances in poultry research since the preceding Congress. In addition, there are a number of other associations related to the poultry industry in one way or another, that hold their meetings during the Congress. The poultry science association, however, is the governing body.

The outstanding feature of the Congress was the exhibits. These were of two kinds, (1) commercial exhibits which included practically everything used by the poultry industry—feed, supplemental feed, incubators, brooders, pens, tractors, trucks, feed grinders and even model kitchens in which poultry products are prepared for the ultimate consumer—and (2) state and national exhibits depicting the poultry industry in the region represented.

The poultry people like themselves pretty well. Wherever one looked, there was reference to the world's this and the world's that, although actually only about 30 nations coöperated, a mere corner of the world. Every record whether made by hen or incubator, a feed or a medicine, was a world's record if not an "all-time world's record."

One would expect in an industry that had grown so rapidly to find the leaders and followers too, alert to the most recent advances in the art and science of their industry, and there was plenty of evidence of this. Blazoned everywhere were the initials R. O. P., which of course mean Record of Performance. The poultry people were way out in front and are still in front of other branches of animal industry in their standard of excellence and recognition. The hen must deliver the goods. The pure



THE MECHANICAL HEN

This mechanism, operated by an electric motor, was a feature of the Massachusetts exhibit. The mechanical heart and the circulation of the blood was quite realistic, but the busy fellows manufacturing an egg had a more difficult task to demonstrate mechanically. Cinema reels and phonographs had a prominent part in several of the state exhibits.

CLEVELAND'S MUNICIPAL AUDITORIUM

The administrative offices of the congress, the scientific and other meetings of the various associations affiliated with the congress, the commercial exhibits and the live poultry exhibit were housed in the ample structure. The exhibits were visited by from 4,000 to 25,000 persons daily.

bred stallion, the dairy cow, the ewe, the sow and most of all, the dog, can still get by on their looks.

On every hand there was evidence of the poultry industry's regard for science. Practically every feed exhibit stressed the research that had gone into building the formula and the results that had been attained with the ration in scientifically controlled experiments. In the exhibit of the American Feed Manufacturers Association conspicuous placards bore such legends as "We serve every American poultryman through scientific standards," and "Laboratory controlled feeds, plus constant research make commercial feeds your best flock insurance," and again, "Commercial feed plants perform an accurate scientific service for American poultry raisers." And, a seal across the hall bore the words, "Stability, Research, Standardization."

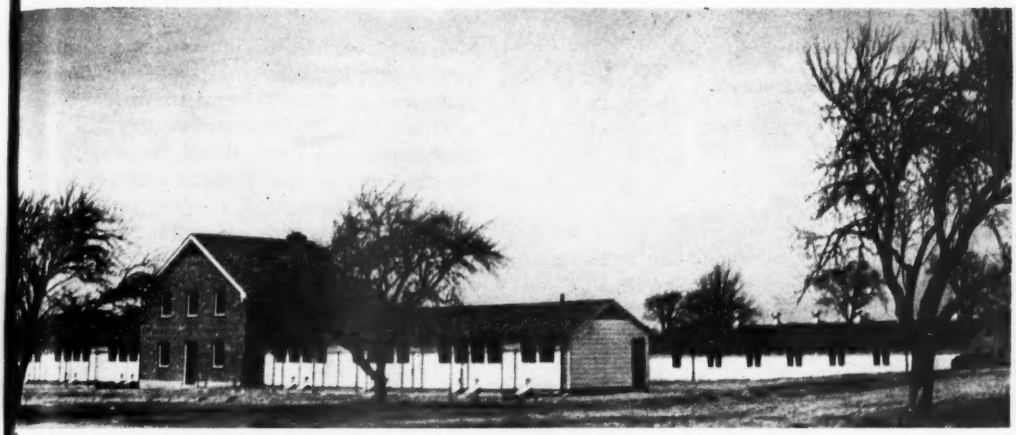
But, the poultryman loves his mumbo jumbo just the same, and right along with the scientific feed exhibits and receiving just as much attention, were intermingled the exhibits of the Doc's—Old Doc Salsbury, Old Doc LeGear, Old Doc Russell and lesser Doc's without number. Each had just the thing for what ails poultry. The Dr. Salsbury Laboratories had the finest and most expensive exhibit in the hall, the most attendants and far the most visitors. It seemed a bit incongruous for the World's Poultry Science Association to father a show in which such a travesty on the science of veterinary medicine was given such a play, and at the same time, invite the representatives of scientific veterinary medicine to participate in their program. A speaker on the scientific program stated that the poultry industry wastes $15,000,000 annually on proprietary remedies that scientific tests have shown to be worthless for the treatment of the conditions for which they are purchased. The statement was not challenged. One wonders if this waste means anything to the poultry science association.

ADMINISTRATION AND CENTRAL LABORATORY BUILDING

The regional poultry research laboratory is located on 50 acres of land donated to the laboratory by the Michigan State College, at the southwest corner of the campus. Elaborate precautions are taken to prevent carrying infection from one laboratory to another and parts of the building assigned to one division of the staff cannot be entered by other members of the staff without special permission. This building is a model of laboratory construction. It is flanked on the east and west by identical sets of buildings, one for the use of the pathology division and the other for the geneticists. There will be no communication between these buildings.

Dedication of Poultry Research Laboratory

The Regional Research Laboratory for the Improvement of Viability in Poultry, established at East Lansing, Michigan, under the provisions of the Bankhead-Jones Act of the Congress, has been referred to a number of times in these pages. The region for which the laboratory is established includes 25 of the north central and north eastern states—Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, Oklahoma, South Dakota, Wisconsin, Connecticut, Delaware, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont and West Virginia.



On each side of the Administration Building, about 50 yards away, are located brooder houses, laying houses and isolation wards.

At the present time the loss from poultry disease in these states is believed to exceed $100,000,000 annually. Half of that loss is thought to be due to fowl paralysis, and the first project undertaken will be a research program for the study and development of control measures for this disease, including neurolymphomatosis, lymphomatosis, leucemoid diseases and sarcomatosis.

The laboratory is assigned to the Bureau of Animal Industry of the U. S. Department of Agriculture and under the immediate leadership of H. C. McPhee of the Animal Husbandry Division, and H. W. Schoening of the Pathological Division of the Bureau.

Although the laboratory began to function last March, it was not dedicated until August 8th, immediately after the close of the World's Poultry Congress in Cleveland. The dedicatory exercises were attended by a number of poultry research workers from foreign countries who had been delegates to the Congress. Altogether more than 175 breeders, technicians, poultry professors and scientific poultry research workers attended the dedication exercises, at which the principal address was made by Dr. John R. Mohler, Chief of the Bureau of Animal Industry.

The present staff of the laboratory consists of Dr. J. Holmes Martin from the University of Kentucky, director; Dr. N. F. Waters from the Iowa State College, senior geneticist, and Dr. C. A. Brandly from the University of Illinois, senior pathologist, together with technicians and care takers. Additions will be made to the scientific staff as the research project develops.

Research will be carried out coöperatively between the central laboratory, the federal Bureau of Animal Industry, the Office of

W. H. ANDREWS, M. R. C. V. S.

Doctor Andrews, of the laboratories of the British department of agriculture and fisheries, Weybridge, England, was a delegate to the VII World's Poultry Congress and also attended the dedicatory exercises of the Regional Research Laboratory for the Improvement of Viability in Poultry at East Lansing immediately after the Congress.

Experiment Stations of the U. S. Department of Agriculture, and the state agricultural experiment stations of the north central and north eastern states.

The immediate objective is to determine the etiologic agent or agents responsible for fowl paralysis, to develop procedures based on etiology, genetics, management and nutrition and to discover prophylactic technique for the prevention or control of this disease. Investigations will be made of various strains of poultry and from the most promising flocks, both resistant and susceptible to fowl paralysis, eggs will be obtained for the further development of resistant and susceptible strains.

* * * *

It is dogs and cats of which we get by far the greatest number for post-mortem examination—over 400 cases in a year out of a total of 500 cases. Amongst these animals we find a very high proportion of neoplasms of one kind and another—actually 24% of the dogs and cats examined were suffering from neoplasms. Nearly a third of these neoplasm cases were epitheliomas (*i. e.*, malignant squamous-celled carcinomas). In the dog, the chief sites were the tonsil and skin. In most of the tonsil cases there were secondary or metastatic tumors in the pharyngeal lymphatic glands and in two or three cases in the lungs. In the majority, however, the metastases were in the pharyngeal lymphatic glands only. In the cat these neoplasms were found mainly in the esophagus and tongue.—*Institute of Animal Pathology* (London).

Hydrogen Peroxide as an Anthelmintic

The day after receiving our copy of the September issue of VETERINARY MEDICINE we had occasion to try the hydrogen peroxide treatment described by Leon F. Whitney (pp. 560-566).

A four-year-old, Scotch terrier was presented with a history of intermittent bloody passages and general unthriftiness. The hair coat was lusterless and scanty, the skin dry and scaly, and there was a bilateral discharge from the eyes. A sugar-dilution, fecal examination indicated a heavy infestation of both hookworms and whipworms.

The animal was immediately given an enema, followed by another 24 hours later, and all food withheld for 36 hours. During this time, until a few hours before the H_2O_2 treatment was employed, the dog had free access to water. One ounce of a 1.5% H_2O_2 solution was administered per os in a gelatin capsule, and 12 ounces of the same solution, lukewarm, was immediately injected per rectum by means of the standard enema tube, inserted eight to 10 inches before allowing the fluid to drain in.

While the enema was being given the animal retched constantly, thus facilitating the upward movement of the rectally introduced solution. On completion of the treatment the dog vomited freely and attempted defecation for 15 minutes. There was no other evidence of distress.

Three hours later the animal was given a good meal, which he ate. A substantial bowel movement followed in approximately four hours. Careful examination of this first movement revealed 125 hookworms and 35 whipworms, Second and third passages brought the total, by actual count, to 216 hookworms and 276 whipworms. More, perhaps, were passed later, after the owner took the dog home.

Since this initial attempt two less severe cases, a Scotch terrier sired by the first patient, and a beagle, have been treated in like manner with the same satisfactory results.

H. J. MAGRANE
W. G. MAGRANE, M.S.C. '40
Mishawaka, Ind.

Encephalomyelitis in Saskatchewan

In an article on encephalomyelitis by Dr. C. Head, Regina, Saskatchewan, which appeared in the July issue of VETERINARY MEDICINE, he plainly states that the findings of research workers in connection with this disease are not acceptable to him. Allow me to say that the views he expressed are not generally held by veterinarians in Saskatchewan; in fact, are unique.

At a conference of veterinarians held in Regina last spring to consider ways and means of handling preventive measures for control of equine encephalomyelitis in this province, there was not a single expression of doubt as to the infectiousness of the disease.

In 1938 equine encephalomyelitis first appeared in various parts of the province on June 28th. Dr. J. S. Fulton, provincial veterinary pathologist, has demonstrated beyond all doubt that equine encephalomyelitis in Saskatchewan is infectious. It attacks horses regardless of age; foals living mainly on the dam's milk were affected as readily as others. Sanitation, food and water supply were not determining factors in producing or preventing the disease. In 1938 most of the horses in the city of Regina were vaccinated. To my knowledge no case developed among those vaccinated. In one unvaccinated herd in this city a fatal case developed; the food and water supply was of the very best, and this animal worked exclusively in the city. The remainder of this herd was promptly vaccinated. The usual experience of veterinarians of Saskatchewan in connection with this disease has been that no new cases develop more than five days following the initial injection of vaccine.

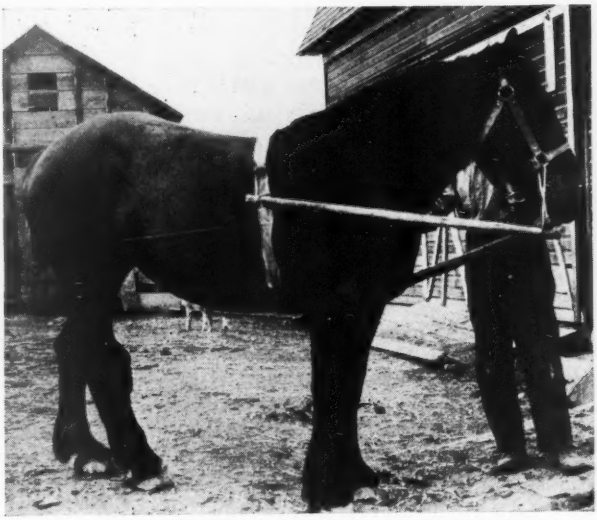
In making mention of his attempts at experimental inoculation, Doctor Head seems not to have taken into consideration that the method used never has proved to be a reliable means of transmitting encephalomyelitis.

In herds where I could vaccinate only a part of the animals—there was quite a shortage of vaccine here last year—encephalomyelitis continued to develop among the unvaccinated ones for two or three weeks. There was a decided abatement about August 15th, long before the hot weather was over and while water in ponds and sloughs was still stagnant. Horses in this district are drinking stagnant water to about the same extent as usual this year, but no encephalomyelitis has been reported so far.

Admittedly botulism and poisoning from damaged food and water do at times occur and necessitate treatment accordingly; but we in Saskatchewan, with the single exception noted, have faith in the findings of research, and in the effectiveness of chick vaccine as a prophylactic for equine encephalomyelitis.

L. L. HEWITT

Regina, Saskatchewan
Canada



To Prevent Gnawing
An efficient apparatus to prevent the patient from biting at a bad wound in the region of the hock.

The American Mule

Of the six members of the genus Equus, only the ass and the horse have been domesticated. The origin of neither is definitely known; but the ass was recorded by Egyptian sculptors as a beast of burden many centuries prior to the Christian era, and long before a record can be found of the domesticated horse.

The ass was used extensively in Biblical times as a means of transportation. It was not allowed to appear at the altar or to be worked with an ox. Absalom rode an ass when he led the rebels against David, his father. Christ made his entry into Jerusalem "meek, and sitting upon an ass."

In a broad sense, the word "mule" denotes the offspring of any two different species, i.e., mixed blood—a mulatto, but ordinarily it is used to mean the hybrid of the mare and jack, the hybrid of a stallion and jennet being called a hinny.

The capacity of this animal to carry heavy loads and draw carriages over mountains or deserts prompted Greek and Roman breeders to develop the mule; performing this work under adverse conditions, it became a real asset, indispensable in war or peace.

For many generations France, Spain, Italy and Portugal have carried on a mule industry, exporting many outstanding jacks to almost every country in the world.

Development of the mule in the United States dates from about 1787, when two jacks were presented to George Washington and placed on his estate at Mount Vernon. One was a present from the King of Spain and the other from the Marquis de Lafayette. These jacks were bred to Washington's Virginian mares. He found the mules sired by the French jack the superior ones. John Clay, in about 1800, imported several valuable Spanish jacks into Kentucky, and these were followed by many others; thus the mule industry developed very rapidly. There are today about 4,600,000 mules in the United States, as compared with some 11,300,000 horses.

In the United States there are five noted breeds of jacks; the Poitan, Catalonian, Maltese, Andalusian and Majorcan. The first two are the most popular. Most of the American jacks of today are a composite of the bloods of the above breeds, emphasizing size, weight, large bone, quality, style and action. A good jack for the production of mules should not be less than 15 hands high, with a straight, strong back, closely coupled and well-muscled over the loin; a long, level rump; a deep body with well-sprung ribs; a broad chest and a large heart girth, including vigor and strength of constitution. The legs should be short and straight, with plenty of bone. The feet should be large and well-shaped. The head should be large, well-proportioned, with the ears long, alert, measuring 33 inches from tip to tip.

Mules are classified according to their use into five classes; the draft mule, the farm mule, the sugar mule, the cotton mule and the miner mule. Then in each of these classes they are graded as choice, good, medium, common and inferior; according to conformation, soundness, quality, condition, style and action. The height varies from 12 to 17½ hands, and weight from 600 to 1600 pounds.

In mule production a good sound mare should be selected, with about one-fourth draft blood and three-fourths light horse blood, similar to the mustang mare. These mares produce wiry, tough, early-maturing mules of high quality that are useful for a wide variety of purposes, especially in the South.

The question is often asked, "Why is the mule sterile?" The horse and ass belong to different, though related species, and their offspring, the mule or hinny, produces spermatozoa or ova with "incompatible" chromosomes. (This is not invariably the case; several cases of fertility in mules and hinnies having been reported, the animals being mated to horses or asses.)

The mule has been bred to his environment, in which he serves better than any other beast of burden. Wherever drudgery and strenuous effort is demanded; if the climate be hot and sultry; if the contour of the land be steep and dangerous; if cour-

age be needed and large demands for endurance be made; if steadiness and surefootedness be required; if the ration is poor or scanty it is the mule that is drafted into

Typical span of farm mules in the middle west.—*Photo courtesy of F. L. Hanna.*

service and his superiority to the other Equidæ under such circumstances is admitted.

The mule is an animal with more undeniable virtues and common sense than any other domestic animal. Treat him kindly and you get a like reception; but treat him harshly and he may treat you to a well-aimed heel. Unlike the horse, he is not prone to abuse himself by overeating, and hence is less likely to suffer digestive disturbances. With his hybrid vigor, he is less susceptible to some diseases than is the horse, although he inherits from his paternal progenitor a high susceptibility to glanders. He lives longer; has fewer ailments; and can endure more work and hardship than the horse.

For these virtues many once prejudiced against him, have come to admire the animal which has no "pride of ancestry or hope of posterity."

FRANK W. JORDAN, K.S.C. '39
Manhattan, Kans.

Carcinomatosis in a Mule

The writer was called recently to see a mule which was staggering around in the barnyard after growing progressively weaker for some weeks. This was a 12-year-old mule furnished by the government to a farmer on the rolls of the Farm Security Administration, and had been selected about one year previously by the county agent. It had done no work for several weeks. Its team mate was in good physical condition. Its ration consisted of ear corn, fed in the shuck, corn fodder and an occasional feed of pea-vine and sorghum hay of good quality. Salt brick was kept in the feed box. The water supply was liberal and of good quality. (This is a soft water section.)

The mule was lying in the sternal position. Its eyes were bright; respirations were accelerated; the pulse was rapid and small. The conjunctiva and the nasal and buccal mucosæ were blanched. The abdomen was enlarged but not tympanitic. The bowel sounds were normal. Auscultation of the flank while irregular pressure was applied

indicated ascites. No anasarca nor stocking of the legs was found, nor were any skin abnormalities seen.

After a light hypodermic dose of digitalin had been administered the animal rose and walked about the barnyard for about five minutes, picking up scraps of corn fodder. Suddenly it staggered and fell, lay still for about 15 minutes, then rose, walked about 50 yards, staggered, fell, and died.

Autopsy revealed a fluid accumulation of 10 or 12 gallons in the peritoneal cavity. There was none in the thoracic cavity. The fluid was red, thin and watery, without the sticky quality of normal blood. It did not coagulate, but soaked into the ground readily, leaving very little residue.

The spleen was about three times normal size, softened, and contained about 30 neoplasms varying in size from a hazelnut to a baseball. These were rather firm, the outer surface being somewhat hemorrhagic, the cortex firm and degenerated, with the approximate color and consistency of boiled egg yolk. There were no signs of calcification, and no pus nor other exudate.

The liver contained about 10 similar growths in the lower half of the principal lobe. No changes were found in any other abdominal organs, and no parasites of any kind were seen. The blood vessels and lymph glands appeared normal.

The lungs were apparently sound, and there were no lesions on the pleura. The heart showed two neoplasms in the musculature similar to those found in the spleen and liver, although they were somewhat more fibrous in character and did not have the degenerated centers found in the others. They were about the size of a lima bean and a medium-sized hickory nut, respectively, and were apparently of recent origin.

The bones and muscles appeared normal.

Diagnosis: Carcinomatosis; anemia (chlorosis) due to destruction of the spleen by the neoplasms.

W. M. BURSON

Athens, Ga.

Urethral Calculus in a Bull

A Hereford breeding bull two years of age which had been straining for two days was admitted to the veterinary clinic of Kansas State College February 10, 1939. The following symptoms were observed: straining, pulsation of the urethra as it turned around the ischial arch, a depressed attitude, and a distended bladder. Temperature, respiration, pulse rate, and conjunctiva were normal, and no other general symptoms were noticed. Diagnosis: urethral calculus.

When water was injected into the urethra with a syringe, only 5cc was admitted. There was therefore a strong possibility that the calculus had lodged anteriorly to the sigmoid flexure, and it was decided to determine the exact location before operation, so that if possible surgical intervention might be so managed as to save the bull for breeding purposes.

The bladder was tapped from the ventral abdominal floor just anterior to the scrotum and about four inches from the median line with a 4½-inch, 20-gauge needle directed medialward; relieving the bladder of most of the urine, but leaving enough so that urination could occur after the operation.

The location of the calculus was determined in the following manner: The penis was massaged and held protruded from the sheath. A No. 20 lead wire (chosen because its flexibility lessened the hazard of damage to the urethra) was inserted into the

This calf was born, appropriately enough, on April Fool's Day.—*W. G. Songer, Lincoln, Kans.*

external orifice and used to explore the urethral tract. The wire was inserted for a distance of about 13 inches before it was obstructed and crepitation felt. It was then removed and used to measure the distance from the urethral orifice to the point of obstruction, along the ventral surface of the sheath.

This region was marked, shaved, disinfected, and anesthetized with 2% procaine. The penis was allowed to be withdrawn into the sheath until anesthesia was complete (about 15 minutes), when it was again protruded. A ventral median incision was made in the marked area and carried through the skin, sheath, and penis up to the urethra, where the calculus was palpated. A small, longitudinal incision was made, and the calculus, about ⅜ inch in diameter, was removed with forceps. The urine immediately began flowing through the opening.

When urination had ceased, the hemorrhage, which had been slight for a tissue as rich in blood supply as the penis, was stopped by means of a pressure pack dipped in liquid bipp, and the skin over the pack was sutured to retain it in place. Three days later, stitches and pack were removed. When the bull was discharged at the end of 10 days, the wound was practically closed, and the animal was urinating in a normal manner.

Although this bull has not been used for breeding to date, the fact that he is urinating in a natural way indicates that no breeding difficulties will be encountered if no undue proliferation of the connective tissue has occurred.

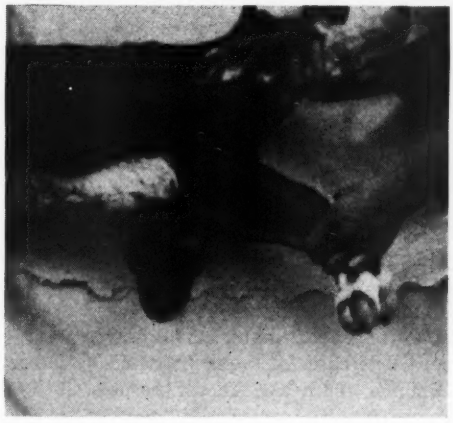
This method of determining the exact location of the calculus before operation when it is anterior to the sigmoid flexure (it is in most cases directly posterior to it) is recommended as a practical measure, since the surgery will then involve only a small area, and there is a better chance that the breeding capacity of the bull will not be destroyed by proliferation of the connective tissue.

SAMUEL S. NEBB

Brooklyn, N. Y.

Ectopia Cordis in a Hereford Calf

Congenital anomalies in the position of the heart are rare. In the condition known as ectopia cordis, the heart with the pericardium is located outside of the thorax. It may be, for example, in the neck or it may protrude through a cleft in the ventral wall of the thorax, the cleft being due to imperfect fusion of the two sides of the body.



The subject of this report was a Hereford calf which was dropped at 5:45 a. m. and died about 4:00 p. m. the same day. The calf could stand but was never able to walk. It appeared normal in every other respect, and was able to take milk twice during the few hours it lived.

As shown in the illustration, the heart was suspended ventrally just under the sternum, and the veins and arteries passed through a round opening two inches in diameter through the center of the sternum.

The heartbeat was regular until 30 minutes before the calf died, when the auricles started to expand.

The case is interesting because it is probably the first ever reported in which the heart was formed entirely outside the body, protected only by the pericardium, but without any skin or other protection.

The case was reported to me by Mr. Charles U. Skouby, science teacher in the high school at Bland, Missouri.

Columbia, Mo. A. J. DURANT

Toxicity of Potassium Cyanide for Swine

By JAMES F. COUCH and HUBERT BUNYEA,
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Washington, D. C.

SWINE are not often poisoned by cyanogenetic plants. This fact has been explained by the assumption that the acidity of the swine stomach is high enough to inhibit the formation of hydrocyanic acid in cyanogenetic materials. However, Fröhner [1] lists cases of swine poisoned by bitter almonds, plum and cherry kernels. It appears from the absence of reports to the contrary that swine are less susceptible to sorghum poisoning than cattle and sheep. However, no data on the lethal and toxic doses of cyanides for swine have been published. It is of interest to know whether swine are more resistant to the cyanides than are ruminants.

During the summer of 1938, there arose an opportunity to obtain some accurate data on the toxicity of cyanides for the pig. A number of animals were made available at the Experiment Station of the Bureau of Animal Industry, at Beltsville, Md. At the time of the experiment these animals were in normal condition. The animals were kept in pens with concrete floors in a large, well-ventilated barn and were allowed an abundance of feed. Measured quantities of potassium cyanide in water solution were administered and the doses were calculated to the hydrocyanic acid equivalent for reference. The potassium cyanide used was of reagent quality. Analysis showed that it contained 95.75% of KCN and all doses were adjusted to 100% for administration.

The results are presented in Table I and the data are broken down in tables II and III. In the last two tables, the terms symptoms, sick, very sick and death are used to designate the severity of the effect. Symptoms denote any effect up to and including mild dypsnea and incoördination; sick refers to cases where dyspnea was severe and the animals collapsed but did not become comatose; the very sick animals became paralyzed or comatose with severe dyspnea. Table II presents the data obtained in cases where the animal did not vomit and table III the data from those cases where emesis took place. Although emesis was delayed in some cases for 40 to 60 minutes after the dose was administered, it was probable that the animal had discharged a portion of the dose and the experiment could not be used to calculate definite dosage. However, since many of the hogs died on small doses even after emesis, it was evident that this factor alone does not prevent fatal poisoning.

Symptoms

The earliest symptoms that appear consist of an increase in the rate of respiration, often accompanied by a spasmodic blinking of the eyes. The respiration rapidly becomes shallow and panting. On the white areas the skin becomes flushed a bright pink. The animal moves about aimlessly as if anxious. Symptoms of incoördination soon appear, the animal staggers, sits on the haunches, or falls on the side. After a short rest the animal rises and recommences a staggering walk about the pen, soon falling again. This rising and falling may be repeated three or four times. The respiration meanwhile becomes more labored, as manifested by the movements of the flank muscles to assist the diaphragm in inflating and deflating the lungs. In aggravated cases there appears a spasmodic respiration acquiring Cheyne-Stokes characteristics. The animal finally collapses, sinks into coma, and dies in respiratory paralysis. During the final period cyanosis is marked.

Many of the animals were salivated. Nausea and vomiting occurred in 48 of the 81 cases, or about 59%. Emesis might take place in four to 10 minutes or might be delayed. In one case the hog vomited one hour after receiving a dose of 1.9mg per kilogram of body weight, from which he

[1] Fröhner, E., 1919. Lehrbuch der Toxikologie für Tierärtzte, Stuttgart, Ed. 4. E. H. Enge (*Jnl. A.V.M.A.* 94:123-124, 1939) has recently reported the poisoning of pigs by wild cherry leaves and roots.

recovered. Seventeen of the hogs that vomited, or about 35%, eventually died. Several of these vomited within 10 minutes after receiving the cyanide. In many cases the hogs vomited several times, one hog vomiting five times within an hour. This animal survived a dose of 3mg per kg.

The principal differences noted in the symptoms shown by hogs and those observed in experiments with cattle and sheep [2] was the delayed appearance of the symptoms in the hogs. The ruminants were quickly affected, the first symptoms of accelerated respiration appearing within three minutes and often within 30 seconds after the cyanide was given. In the hog experiments one animal was affected in two minutes after receiving a dose of 5.1mg per kg and another in 2.5 minutes after a dose of 2.4mg per kg and a third in 2.5 minutes after a dose of 2.5mg per kg. In the other cases symptoms usually appeared within 10 minutes, but in 16 cases they appeared between 10 and 20 minutes and in three cases did not appear until 28, 42 and 47 minutes, respectively, after the dose had been given. The ruminants usually collapsed in one to five minutes after receiving the cyanide. This symptom was much delayed in the hog cases. While one animal collapsed in 2.5 minutes and three others in 3.5 minutes, all on large doses, most swine required much longer, and in 14 cases the time was between 20 and 38 minutes after the cyanide had been given.

In several cases hogs recovered after becoming apparently moribund, the respiration slowing to two to four shallow inspirations per minute. Spontaneous recovery of sheep or cattle in a similar stage of intoxication was never observed.

Death occurred in from 20 minutes to several hours, a number of animals dying during the night, at least six to seven hours after receiving the cyanide. Thirteen animals died in 20 to 60 minutes, five died in one to two hours, and 11 after a longer period. With sheep and cattle the average time to death was 25 to 35 minutes, with six minutes the minimum and 90 the maximum observed.

[2] Clawson, A. B., J. F. Couch, and H. Bunyea, 1934. *Jnl. Wash. Acad. Sci.* 24:369-395, 528-532; 1935, 25:57-59, 272-276, 357-361.

As is usual in cyanide poisoning those animals that survived made a rapid recovery and were apparently normal on the day following the experiment.

Toxic and Lethal Doses

In the cases where there was no emesis, all of the animals that received 3.4mg per kg HCN equivalent died. Four animals died on doses smaller than this, one succumbing to the low dose of 1.7mg per kg although one animal recovered from a higher dose. The lethal range for the hog may therefore be set at 2.4 to 3.4mg of HCN per kg. In the same set of cases the smallest dose that produced symptoms was 0.65mg per kg although four animals given somewhat larger doses showed no symptoms. Severe symptoms were shown by animals that received 0.86 to 1.9mg per kg although a higher dose, 3.1mg per kg, produced merely symptoms. The lethal dose of HCN for sheep was found to be nearly 2.3mg per kg and for cattle, 2.0mg.[3] The indications are that the hog is somewhat more resistant to lethal effects than sheep or cattle, even aside from his ability to vomit up the poison. However, symptoms were observed on smaller doses in hogs than in sheep, 0.99mg, and cattle, 0.88mg per kg. The course of the poisoning in hogs indicated a marked resistance as compared with the ruminants.

Effect of Emesis

In those cases where the animal rejected part of the dose by emesis the lethal range cannot be accurately estimated. Eleven animals died on doses ranging from 2.2 to 3.5mg per kg and 13 animals survived doses in this range after emesis. Six animals survived the higher doses of 4.3 to 5.4mg per kg after emesis. In all, 19 animals survived probable lethal doses mainly through getting rid of a portion of the poison. The smallest dose that produced symptoms including emesis was 0.84mg per kg in the case of pig No. 29. The symptoms were more severe than those shown by pig No. 28 that did not vomit on a slightly higher

[3] Clawson, Bunyea, and Couch, 1934. *Jnl. Wash. Acad. Sci.* 24:369-385. More recent experiments indicate that the m.l.d. for cattle may be higher than this figure under certain conditions.

dose, 0.86mg per kg. Certain pigs appeared to vomit more readily than others. Pig No. 5 was given the cyanide on nine different occasions and vomited on seven of them. Doses of 0.24 and 0.84mg per kg did not provoke emesis in this case, the lowest

TABLE I. HOGS GIVEN KCN SOLUTION BY MOUTH

Date 1938	Hog No.	Weight Kilograms	Dose mg. per kg.*	Effect	Minutes from administration to			
					Symptoms	Emesis	Collapse	Death
Aug. 3	1	183.7	1.9	Sick; emesis	1.25	60	26	
" "	2	197.3	2.7	Very sick; emesis	14.5	14½	30	
" "	3	208.6	5.1	Death	2.		2½	20
" "	5	167.8	2.4	Sick; emesis	2.5	9½	‡	
" "	7	152.9	3.5	Death	5		10½	120
" "	8	215.4	4.1	Death; emesis	5	8	9	43
" "	11	181.4	3.6	Death	3.5		3½	41
Aug. 8	12	215.4	2.8	Very sick; emesis	6.5	6.5	24	
" "	13	208.8	3.4	Death; emesis	3	10	4	†
" "	14	224.5	3.1	Emesis; death	3.5	8.5	22½	60.5
" "	15	180.1	3.4	Very sick; emesis	4	3	‡	
" "	18	176.8	3.5	Emesis; death	2.5	7	3½	26
" "	19	260.8	3.4	Death	3		23	26
" "	20	249.4	3.5	Death	3		7	27
Aug. 11	1	183.7	2.9	Sl. sick; emesis	7	7		
" "	4	199.5	2.6	Death; emesis	2	6	6½	70
" "	5	167.8	3.1	Sl. sick; emesis	3	4		
" "	6	192.7	2.5	Death	2.5		6	26
" "	10	254.	1.7	Death	5.5		6	†
" "	12	215.4	3.3	Death; emesis	4	7	4	†
" "	15	180.1	3.5	Emesis; death	11	15	11	†
" "	17	229.	3.4	Death	4		7	31
Aug. 17	22	193.2	2.7	Emesis; death	5	11	26	63
" "	23	226.7	2.7	Death	10		21	40
" "	24	204.1	2.7	Death; emesis	7	8	16	54
" "	25	351.5	3.5	Death	5		‡	62
" "	26	147.5	2.4	Death	7.5		10	58
" "	32	120.2	2.2	Emesis; death	8	13	24	†
" "	33	124.7	2.8	Emesis; death	7	16	27	†
" "	34	124.7	2.8	V. sick; emesis	7	7	27	
" "	35	77.1	2.3	Emesis; death	9	16	20	†
Aug. 22	21	260.8	1.3	Sick	4		‡	
" "	28	163.3	0.86	Sick	3		‡	
" "	29	136.1	0.84	Emesis; very sick	10	11	20	
" "	30	161.	0.87	Sick	4		38	
" "	31	140.6	0.91	Sl. sick; emesis	14	14		
" "	34	124.7	1.1	Depressed	11			
" "	5	167.8	1.1	Sick; emesis	7	7	14	
" "	1	183.7	0.86	Sick; emesis	9	9	‡	
Sept. 9	28	163.3	0.27	None				
" "	29	136.1	0.32	"				
" "	30	161.	0.27	"				
" "	31	140.6	0.31	"				
" "	34	124.7	0.35	"				
" "	21	260.8	1.3	Sick; emesis	9	10	‡	
" "	1	183.7	0.26	None				
" "	5	167.8	0.24	"				
Sept. 12	1	183.7	0.72	"				
" "	5	167.8	0.84	"				
" "	21	260.8	2.0	Emesis; sick	7	14	‡	
" "	28	163.3	0.81	None				
" "	29	136.1	0.65	Sl. depressed	20			
" "	30	161.	0.66	Sl. depressed	28			
" "	31	140.6	0.75	Resp. accelerated	11			
" "	34	124.7	0.80	Depressed; resp. symp.	23			
Sept. 15	1	183.7	1.9	Sick; emesis	6	6	‡	
" "	5	167.8	2.1	Sick; emesis	12	29	‡	
" "	21	260.8	3.0	V. sick; emesis	10	41	‡	
" "	28	163.3	1.6	Sick; emesis	8	8	16	
" "	29	136.1	1.3	Sick; emesis	26	28	‡	
" "	30	161.	1.0	Sl. symptoms	13			
" "	31	140.6	0.93	None				
" "	34	124.7	2.1	Sick; emesis	42	42	‡	
Sept. 21	1	183.7	3.8	Emesis; death	14	19	14	†
" "	5	167.3	3.1	Sick; emesis	8	14	8	
" "	21	260.8	4.7	Death	4		4	24
" "	28	163.3	3.2	V. sick; emesis	7	7	12	
" "	29	136.1	3.9	Emesis; death	4	7	4	†
" "	30	161.	3.3	V. sick; emesis	19	27	36	
" "	31	140.6	3.1	Weak	47			
" "	34	124.7	3.5	Sick; emesis	21.5	21.5	‡	
Sept. 26	5	167.8	4.2	Sick; emesis	12	12	‡	
" "	28	163.3	4.3	V. sick; emesis	3.5	5.5	3½	
" "	30	161.	4.3	V. sick; emesis	7	7	‡	
" "	31	140.6	4.4	V. sick; emesis	9	9	‡	
" "	34	124.7	3.5	Weak; not very sick; emesis	4	10		
Oct. 3	5	167.8	5.2	Emesis; v. sick	12	12	‡	
" "	28	163.3	5.4	Emesis; death	11	18	17	†
" "	30	161.	5.4	V. sick; emesis	3	7	3	
" "	31	140.6	6.2	Emesis; death	5	8.5	8	22
" "	34	124.7	7.0	Emesis; death	3	6	6	†

* Calculated to HCN. † During the night. ‡ Animal lay down voluntarily. Time of onset of collapse not determinable.

emetic dose being 1.1. The time to emesis with this animal varied from four to 29 minutes after administration of the cyanide, the average being 14.6 minutes. This animal survived the comparatively large doses of 4.2 and 5.2mg per kg, vomiting in each case in 12 minutes.

TABLE II. EXPERIMENTS WHERE THERE WAS NO EMESIS
Dose and effect.

Hog No.	Death mg per kg	Sick mg per kg	Symptoms mg per kg	Not Sick mg per kg
3	5.1			
21	4.7			
11	3.6			
7, 20, 25	3.5 3.5 3.5			
19, 17	3.4 3.4			
31			3.1	
23	2.7			
6	2.5			
26	2.4			
10	1.7			
21		1.3		
34			1.1	
30			1.0	
31				0.93
30		0.87		
28		0.86		
5				0.84
28				0.81
34			0.8	
31			0.75	
1				0.72
30			0.66	
29			0.65	
34				0.35
29				0.32
31				0.31
28, 30				0.27 0.27
1				0.26
5				0.24

The data furnish little evidence either of increased susceptibility or of the establishment of tolerance. Those animals that received several doses for the most part vomited the toxic doses making accurate calculations impossible. Pig No. 1 died on a dose of 3.8mg per kg with emesis and after surviving six smaller doses given during the previous seven weeks. Pig No. 29 died on 3.9mg per kg after vomiting. This animal had received four smaller doses during the previous four weeks. However, pigs Nos. 5, 28, 30, and 31 survived doses ranging from 4.2 to 4.6mg per kg after vomiting. These animals had previously received five to seven doses of the cyanide. Pigs Nos. 28, 31, and 34 died on large doses of 5.4, 6.2, and 7.0mg per kg respectively although emesis took place. On the other hand pigs Nos. 5 and 30 survived doses of 5.2 and 5.4mg per kg after emesis in 12 and seven minutes respectively. Pig No. 28 that died on a dose of 5.4 vomited in 18 minutes.

Acknowledgement

The writers wish to acknowledge their indebtedness to Dr. J. W. Dollahite, who made certain of the feedings, and to Drs. W. S. Gochenour and C. G. Grey, who made the experimental animals available.

TABLE III. EXPERIMENTS WHERE THERE WAS EMESIS
Dose and effect.

Hog No.	Death mg per kg	V. Sick mg per kg	Sick mg per kg	Symptoms mg per kg
34	7.0			
31	6.2			
28, 30	5.4	5.4		
5		5.2		
31		4.4		
28, 30		4.3 4.3		
5			4.2	
8	4.1			
29	3.9			
1	3.8			
18,15,34,34	3.5 3.5	3.5	3.5	
13, 15	3.4	3.4		
12, 30	3.3	3.3		
28		3.2		
14, 5, 5	3.1		3.1	3.1
21		3.0		
1				2.9
33, 12, 34	2.8	2.8 2.8		
24, 22, 2	2.7 2.7	2.7		
4	2.6			
5			2.4	
35	2.3			
32	2.2			
5, 34			2.1 2.1	
21			2.0	
1, 1			1.9 1.9	
28			1.6	
21, 29			1.3 1.3	
5			1.1	
31				0.91
1			0.86	
29		0.84		

Summary

The pig is somewhat more resistant to cyanide poisoning than cattle and sheep. The minimal lethal dose when there is no emesis ranges from 2.4 to 3.4mg per kg calculated as hydrocyanic acid and administered as potassium cyanide. In 59% of the cases the animals vomited part of the dose. Eight survived much higher doses than the above. Toxic effects were obtained with a dose of 0.65mg per kg and severe symptoms with doses of 0.86 to 1.9mg per kg. The smallest emetic dose was 0.84.

The symptoms observed were similar to those seen in cattle and sheep but were much delayed. Pigs appear to be much more slowly affected than ruminants. This fact, combined with the ease with which the pigs can empty the stomach, probably accounts for the relative immunity of the hog to spontaneous poisoning from cyanogenetic plants.

There was no indication of the establishment of tolerance or of increased susceptibility.

Owning a Dog Should Be a Pleasure, Not a Trial

By JOHN W. PATTON,
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THREE-FOURTHS of our dogs suffer to a greater or less degree from nutritional inadequacy—or malnutrition. This is a comment we have been making for years and further study only strengthens our conviction in this respect. The causes for this sad condition are many and varied, but the responsibility belongs to the rank and file of the dog food manufacturers and the dog-food-buying public.

The average dog food manufacturer has relatively no knowledge of the nutritional requirements of the dog he hopes to feed. His product is usually made to satisfy the palate of the dog while ignoring the simplest of his bodily requirements. These belly-filler dog foods do little more than relieve the sensation of hunger by distending the stomach. It was to describe these classes of dog foods that the phrase "Starving to death on a full stomach" was coined.

Among the manufacturers of dog food, as in any other line of endeavor, there are both conscientious and unscrupulous operators. The average manufacturer of dog food makes what the public will buy. The dog-food-buying public has done much to make the manufacture and sale of dog food a racket. By its support it has popularized the 5c can of dog food, notwithstanding the fact that no can of dog food made to sell for five cents can come anywhere near providing the nutritional requirements of dogs. The man who makes such food knows this if the user does not. An old axiom states: "You get what you pay for." In canned dog food, 5c pays for malnutrition.

The manufacturers of standard-priced brands of dog foods have reduced their price and most such manufacturers are making a better dog food than at any time in their history. Conversely, the manufacturer of cheap dog foods, especially canned dog foods, have hit bottom as scavengers in the vegetable and animal kingdom in order to secure materials which can be used to keep production in pace with the increasing demands for cheap dog food. On today's market the best buy is standard-priced dog foods—not cheap dog foods.

Few dog owners know the nutritive requirements of dogs or how to provide them. Fewer still have any conception of the difficulty of meeting these requirements and selling a commercial dog food in spite of the dog owner's predilection for a five-cent can. Ignorance on the part of the purchaser is the basis upon which the sale of cheap dog food exists. If the buying public would only employ common sense and reason in the purchase of food for its dogs it could do much to protect its pocketbook and at the same time provide its dogs with badly-needed nutrition, for there are good foods available. Certain leading firms among the manufacturers of standard priced dog foods have expended large sums on research in canine nutrition and have developed adequate foods. Furthermore they have the scientific data to prove it.

■ Some knowingly buy cheap dog foods, use them as a base and attempt to balance the diet from other sources. This is a practical impossibility. To properly balance the diet, when using cheap foods as a base, it is (1) necessary to know the nutritive value of the base dog food. Inasmuch as no manufacturer of cheap dog food will ever dare to tell (truthfully) what it contains, the safest course to pursue is to attempt to ascertain this information from independent study. (2) When, and if, the nutritive value of the base food is learned, it is then necessary to figure out the dog's nutritive requirements and calculate the quantity of nutrient factors required to effect a balance. The procedure for determining the nutritive value of the base dog food would be somewhat as follows:

1. Disregard the label* and the advertising claims. These are valueless as a declaration of the contents and an unsafe guide for nutritional expectancy.

2. Make a vitamin assay for at least vitamins A, B_1, B_2, B_4, B_6, D and E.

3. Institute growth studies and calculate grams gain in weight per grams of food consumed. Study the average gain in weight and compare with the standard growth curve. These studies should be carried out for at least a period of 90 days under controlled laboratory conditions. Due to the fact that certain deficiencies, which will affect the balance, are best determined by regenerative studies, it is safer that these studies be carried into the third generation.

4. Make a complete chemical analysis of the base food, paying particular attention to the quantitative value of the proteins and fats. If the base food is canned, find out—if possible—if the protein is from soy bean meal or muscle tissue. If the base food is a dry food and especially if it is a chicken mash or uncooked type, be most careful in determining its coefficient of digestibility for the dog. Make a study of the mineral content and calculate the calcium-phosphorus ratio.

It is not what the dog eats but what it assimilates that nourishes its body. In cheap dog foods there are many ingredients present which the dog cannot break down to an absorbable point. These, obviously, have no place in a dog food for they pass out undigested and the energy expended in an attempt to assimilate them is wasted. There are other pertinent tests and studies which assist in a determination of the amount of balance required but those stated above will prove quite helpful.

5. Next, calculate the caloric and vitamin intake requirements of the dog, or dogs, that are to be fed on the food, in accordance with the age, weight, sex, metabolic rate and conditions of life; that is, the amount of work performed, exercise taken, exposure endured, etc. Having ascertained these values, deduct from them the values obtained from the cheap dog food base and the difference is the amount of balance required to bring the dog food up to the level of a standard dog food.

■ The foregoing procedures require the expenditure of a lot of time and money on a 5c can of dog food, especially when it is necessary to go through the entire procedure on every purchase, because cheap dog foods are variable, made from the materials available at the moment, and no two lots are the same.

It is obvious that the self-proclaimed dog food expert who buys a base with the serious intent of supplementing to construct a balanced diet is fooling himself far more than anyone else. Few dog owners have the training and facilities to accomplish this task even if it were economically practical, which it is not. The average dog feeder harbors more superstitions and fallacies along nutritional lines than the average cattle, horse, poultry or swine feeder. Given two dogs and in his own mind he immediately becomes an authority on canine nutritional matters.

■ The rank and file of manufacturers of cheap dog foods are indifferent; dog owners are well-meaning but credulous; and 75% of our dogs are undernourished. What is the cause? We believe it resides in the fact that the dog is an animal of high sentimental value and low economic importance. The economic importance of farm animals has resulted in a great deal of research in their behalf. The poultry breeder, for instance, can write to the Federal Government or any one of the state agricultural colleges and get voluminous data on almost any problem connected with poultry production and marketing. A similar service is available to other livestock owners. Like research on dogs is not available to dog owners. There are no federal or state funds available to delve into matters concerning the dog's health and well-being. What information we have on the subject has come to us through (1) medical research in

*The time allowed manufacturers to comply with the label requirements of the new Food, Drug and Cosmetic Act has been extended from June 25, 1939, to January 1, 1940, so the label is as yet no more truthful than the manufacturer or his advertising agency who writes it.

which the dog has been used to elucidate problems concerning man; (2) the veterinary profession, which has contributed much to the prevention of disease and the better treatment of the dog's bodily ills, and (3) the conscientious manufacturers of dog foods, who by independent research have increased the available knowledge on canine nutrition. These are the fountainheads from which has sprung most of the scientific information we possess on matters pertinent to the dog. There are other agencies, of course, such as those engaged in humane activities which have bettered the dog's position in life; however, these are palliative in removing effects rather than exploring or advancing our knowledge on the cause of matters affecting the health and well-being of the dog.

This means, therefore, that the breeding and care of farm animals is based upon a far more scientific foundation than the propagation and feeding of dogs. In the case of farm animals, scientific studies have replaced much of the superstition and fallacy. In the dog, because of lack of scientific data, superstition, fallacy and private opinion rule, and these are in no sphere more evident than in the feeding.

■ The results of feeding an improper diet are many and varied, ranging from mere indisposition to actual loss of life. That dogs often die as a result of improper diet is common knowledge among veterinarians. It is not so easily recognized, however, that these same inadequacies, in less degree or over a shorter period, affect the health of the dog; particularly its resistance to disease and its capacity for convalescence. In these boderline cases of malnutrition the important part nutrition plays in animal health is obscured.

It is well for the dog owner to keep in mind the fact that the healthy body possesses a huge tolerance for most diseases, and that the healthy body is one which is in a good state of nutritional equilibrium. I do not mean to convey the impression that adequate nutrition is a panacea for all the ills to which a dog may fall heir, or that an adequately nourished animal will not contract some infectious diseases. However, the well-nourished dog can be expected to be free from nutritional deficiency diseases, and can be expected to offer more resistance to disease in general and to possess a high recuperative power.

Research by this writer has demonstrated that it is possible to rear dogs practically from birth to old age without their ever experiencing a sick day. In a series of experiments several litters of puppies were fed from weaning time to seven months of age on a diet which was calculated to provide qualitatively and quantitatively their daily nutritional requirements. At the same time a similar group of puppies was fed on a diet calculated to provide the same level of intake, quantitatively *or* qualitatively (but not both). This second group grew into dogs lacking the robust constitution of the former and later proved more subject to disease than the first group.

In other studies we have found—as would be expected—that it is necessary to start early, the earlier the better, to nourish puppies. In our experimental work it made a great deal of difference in the results if we conducted an experiment with puppies acquired at weaning time and of unknown nutritional history or if we raised our own puppies from bitches whose diet was calculated to maintain a good state of nutritional equilibrium prior, during and following gestation. Many breeders and dog owners wait until weaning time to start nourishing their litter of puppies without realizing either that many puppies at this stage of development are destined to die prematurely because of previous nutritional deficiencies or that others less seriously affected are in such a poor state of nutritional equilibrium as to render them fit subjects for malformations and deficiency disorders, and suitable hosts for the successful invasion of many and sundry disease-producing organisms.

We have had litters born that no conceivable measures could possibly save from an early death because of the dam's diet. The puppies were already doomed at weaning time—yes, at birth they were destined

to premature death because of nutritional deficiencies of the bitch that bore them.

This is not altogether a new thought—that animals may be born destined to die prematurely. The geneticist is familiar with what he calls "lethel factors," that make survival for more than a short period impossible. A number of species of plants—especially cereals—at times produce strains with a deficiency in chlorophyl production, and those so affected die soon after germination. In the animal kingdom we have still further evidence of the operation of lethal factors along genetic lines. There are Drosophila that are doomed to premature death from the moment of conception and the well-known instance of the classical homozygous yellow mouse that cannot survive because of lethal factors. It is not so fully appreciated, however, that animals, too, may be born with nutritional deficiencies that just as inevitably doom them to premature death.

However, these drastic deficiencies are not on the whole important to the dog owner. In the main they are exaggerated aspects of the matter that cure themselves by removing the dog after a few days, or at most a few weeks. Their chief interest lies in the fact that they do occur naturally and they can be produced experimentally with mathematical certainty and clocklike regularity. It is the milder, sub-lethal nutritional deficiencies from which most dogs suffer; those deficiencies that stunt the growth, make the dog irascible when he should be friendly, stupid when he should be alert, sluggish when he should be playful, delicate when he should be robust, and prepare in his body a fertile field for parasites and bacteria that he could normally ignore—that, in short, make what is potentially a satisfactory friend and companion into a subject requiring more or less constant pampering and nursing. These borderline deficiencies we shall not overcome until the dog-owning public gets over its obsession that a 5c can of dog food is fit for a dog or that a good chicken mash can be mixed with water and do more than fill a dog's belly and satisfy its appetite.

Spread of Canine Distemper

I have had a good deal of canine distemper in my practice the past season. A peculiarity of it has been that many cases have had no history of contact with any dog from which the infection could have been acquired. In fact, a few cases can be said positively to have had no possible contact with any dog.

Many New York city people have country homes in this section in which they spend the summer. They drive into town on Saturdays for shopping; often taking their dogs in the car but not letting them out while in town. Street dogs attracted to the car by these animals of course urinate on the wheels and tires. The first thing the owners' dogs do when the cars reach home and they are released is to smell around these wheels. This may be a source of infection and seems the most plausible explanation of these definitely "no contact" cases. I believe the owners of dogs should be advised of this hazard to the health of their pets.

A. N. TOWNER

Towners, N. Y.

Comment: In his very thorough investigation of canine distemper in foxes and dogs Green[1] found the distemper inclusion bodies very numerous in the epithelium lining the bladder. In a personal communication he has stated that the elimination of the Carré virus from an infected animal is chiefly through the urine and that probably that excretion is the chief vehicle in the spread of the infection.

* * * *

The Michigan assembly last winter passed with little opposition a bill known as the "Kill a dog and get $5.00" bill. It provided that anyone killing a dog would receive $5.00 from the county treasurer upon making an affidavit that he suspected the dog of killing sheep. The bill was defeated in the state senate because of its probably high cost for the $5.00 rewards.

[1] Green, R. G. and C. A. Evans, 1939. Rapid Diagnosis of Canine Distemper. *The Cornell Veterinarian*, 29:1, pp. 35-40.

Paralysis of the Jaw in a Dog

May 20, 1939, a house call was made to examine a five-year-old, male, English setter which had that morning evidenced a paralysis of the lower jaw and was unable to eat or drink. The owner thought that there was a little change in the facial expression. The only time the dog had been out was during hunting trips, under the owner's constant surveillance.

Examined through a wire fence, the patient appeared restless, and his lower jaw was dropped, saliva hanging in long strands from it. Frequent attempts to drink water were made, but the amount of liquid in the water pan was only increased by deposition of saliva. No other classical symptoms of rabies were evidenced, nor was there any evidence of foreign body obstruction, fracture or bulbar paralysis.

In view of the serious possibilities of the case, despite the fact that rabies had not been reported in the community for five years, the owner was advised to confine the animal closely, for which procedure he had good facilities, and to be on the alert for other symptoms, such as change of voice, chasing moving objects, staring expression, and weakness of the hind parts. Routinely, the health authorities and the owner's physician were advised of the circumstances.

From the second day on, steady improvement in the dog's condition was noted. He was soon able to eat by seizing his feed and throwing it into his pharynx with a quick jerk of the head. No further symptoms were noted, and ten days after the first sign of illness, a manual examination of the mouth and throat was made, using rubber gloves. The findings were negative, with the exception of a lighter-than-normal mucosa, and the dog appeared normal.

Whatever the conjecture as to cause, the point to be brought out is that a diagnosis of rabies should depend on more than paralysis of the jaw and pharynx. While the owner and the authorities should be advised of the possibility of rabies, and the animal fed and watered in strict quarantine, a definite diagnosis should be withheld pending observation of the course of the condition within the established time limits of true rabies.

MATTHEW J. EGGERT

Roanoke, Va.

McKENNA SUCCUMBS TO LONG ILLNESS

Dr. John F. McKenna at his Desk in the Ambassador Dog & Cat Hospital, Los Angeles, Calif. Doctor McKenna, leading small animal practitioner and one of the most widely known veterinarians in the country, died August 14th at Johns Hopkins Hospital in Baltimore. He had been ailing three years from carcinoma of thoracic organs, which caused his death. He is survived by his widow, a son and a daughter.